THE
LARYNGOSCOPE.

VOL. L SEPTEMBER, 1940. No. 9

OTOGENIC COMPLICATIONS. A RESUME AND DISCUSSION OF THE LITERATURE FOR 1939.

DR. LYMAN G. RICHARDS, Boston.

In a survey of the past year's literature dealing with otitic complications, the same system of subdivision will be followed as that of our previous review. Thus, the extension of infection originating in the middle ear and mastoid process will be classified according to involvement of: *1.* the meninges and subarachnoid spaces; *2.* the labyrinth; *3.* brain tissues, either cerebellar or cerebral; *4.* the lateral sinus and its subdivision; and *5.* the petrosal pyramid. Mention should first be made, however, of several excellent articles less amenable to this classification which deal with the application of major therapeutic measures, including chemotherapy, to otitic sepsis in general, without respect to the particular anatomical region involved.

In a symposium on the care of the patient following operation for sepsis of otitic origin, Grove[1] stresses the importance of competent and specialized nursing and of the administration of the type of food most likely to sustain the patient's resistance. In addition, he gives excellent advice as to the parenteral administration of fluids for purposes of combating any tendency to dehydration, acidosis and vomiting. He advises frequent examination of the urine for the detection of acetone and diacetic acid, and for injection states a preference for a 5 per cent solution of dextrose in physiological sodium chloride, using three to four quarts daily for an adult, and two to three quarts for a child. A 5 per cent solution is preferable to one of 10 per cent since the latter, given in

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Sept. 1, 1940.

large amounts, tends to spill over and to be recovered from the urine. Dextrose in saline solution may be alternated with dextrose in triple distilled water to prevent edema due to excessive chloride.

Sutherland,[2] in a discussion of the value of blood transfusion in otitic sepsis, feels this to be of value in counteracting the following conditions: *1.* hemorrhage, traumatic, secondary to local disease or secondary to operation; *2.* shock, either hemolytic or surgical; *3.* anemia, complicating a local disease; *4.* a progressive fall in concentration of hemoglobin; *5.* toxemia complicating mastoiditis or sinus thrombosis; *6.* debilitation, with or without secondary anemia; *7.* a loss of plasma protein associated with acute anhydremia, especially in infancy and childhood; and *8.* acute hemolytic anemia secondary to the use of sulfanilamide, for which blood transfusion should be given until the blood level is restored. The general therapeutic value of blood transfusions, he feels, depends on the fact that they supply serum and plasma proteins which aid in restoring blood volume and water balance within the vessels, thus diluting toxin products in the blood. Second, they provide an immediately available supply of oxygen carriers. Third, they furnish a quantity of fresh leukocytes for the elaboration of bacterial enzymes, opsonins, agglutinins and bactericidal plasma or serum, which render the bacteria more readily phagocytosed. Fourth, they stimulate the hemopoietic centres, thus aiding in the formation of white and red cells, and, finally, they support hepatic function which is essential to blood and hemopoietic efficiency.

As specific methods of transfusion, Sutherland recommends either whole unmodified blood by direct transfusion, citrated or modified blood by indirect transfusion, specifically immunized blood, nonspecifically immunized blood, and immunized blood and blood serum from convalescent donors. If possible, it is still better, as advocated by Gill, to secure blood from patients who have survived infections similar to those affecting the patient. As origins of unfavorable reaction attending transfusion are listed: *1.* the use of specifically incompatible blood; *2.* the presence in the donor's blood serum of nonspecific protein components and other substances which tend to produce allergic phenomena; *3.* the use of citrate for transfusion; *4.* incipient coagulative changes in the transfused

blood; *5.* foreign matter derived from the apparatus; *6.* introduction of too large an amount of blood, overburdening the patient's circulation with possible pulmonary and cerebral edema; and *7.* indiscriminate use of a universal donor whose agglutinins are of too high a titre.

In an excellent article on the use of chemotherapy in otitic sepsis, Galloway[3] enunciates the following principles. If chemotherapy has not been given before operation, and the patient presents adequate indications for intervention, with a presumably susceptible organism, the drug should be given to shorten convalescence, avoid complications and diminish mortality. Masking of more serious trouble must always be borne in mind. If, in spite of adequate chemotherapy, operation becomes necessary, further administration of the drug will not be required if it is possible to remove the disease and institute satisfactory drainage. Usually the drug will have proven to be of some benefit and failure may have been due to too late or improper administration. Subsequent resumption of the drug may seem desirable if it is well borne. If toxic symptoms occur, especially severe ones, and surgical intervention seems sufficient the drug may be stopped. In the presence of meningitis, chemotherapy will probably have to be continued and toxic symptoms met as they arise by such agents as blood transfusion. Sinus thrombosis can usually be dealt with successfully without the drug but here, also, it seems to hasten cure and prevent the danger of local and generalized extension of the sepsis. With petrositis, if one is not sure of adequate drainage and unblocking, sulfanilamide may be withheld for further study unless there are signs of meningeal involvement or unless complete operative removal seems impractical. In the presence of labyrinthitis it is now possible to adopt a more conservative surgical attitude and place considerable reliance on chemotherapy, provided that the patient is under almost hourly observation and is not showing signs of meningeal irritation. Abscess of the brain should, of course, be treated surgically, but chemotherapy, except in the case of severe toxicity, should be used for additional protection against extension of the infection, and particularly in the presence of secondary meningitis.

An interesting summary of the classical clinical problems to be dealt with in otitic complications is presented by Juers,[4]

who reports eight cases of petrositis with six recoveries by operation, and two cases of labyrinthitis, in one of which the patient was cured without labyrinthectomy and one in which the patient died in spite of this operation. He also records six cases of brain abscess, with two recoveries and four fatalities, the abscess being in two instances in the cerebellum, and in four in the cerebrum. It must be acknowledged that these complications were encountered before the active use of sulfanilamide, but the author feels, in spite of this, that a major number of the fatalities were due to the neglect of the chronic otitis media, in which surgical delay is most important and in which earlier operative procedures might have prevented such an outcome.

An interesting testimony to the increased interest on the part of pediatricians in problems of otitic sepsis is presented in an article by Aries,[5] who summarizes the proper pediatric treatment in these conditions and stresses the value of rest, intelligent feeding, the use of intramuscular blood injections or transfusion, particularly of immunotransfusions. He feels that the use of sulfanilamide in meningitis has rendered the need for daily lumbar punctures less urgent, and also particularly the need of intraspinal medication.

In a comprehensive article on otitic infection caused by pneumococcus type III, Maybaum[6] summarizes his experience with complications incident to infections by this particular organism. He feels that lesions due to pneumococcus type III are more prone to give rise to intracranial complications than infections of streptococcic origin and that of these, meningitis is most common. It may occur early in the first week of the otitis or more commonly late, after an insidious course without evidence of extensive temporal bone disease. Extension of the process to the petrosa is not uncommon. In Maybaum's group of 13 instances of this complication, the diagnosis was made clinically in eight, and in the remaining five, with no physical signs or symptoms, by postmortem or histological examination. He feels that thrombophlebitis of the sigmoid sinus and jugular bulb is a comparatively uncommon complication with infection of type III pneumococcus, occurring in his series in only three cases. The clinical course of such pneumococcic thrombophlebitis is not infrequently atypical and may be asymptomatic throughout, the diagnosis being

made only by recovering the organism from the blood. Labyrinthitis complicated the otitis in three instances, in two of which the diagnosis was made clinically and in the third from histological examination. There were two abscesses of the temporal lobe due to the pneumococcus, the complication occurring as a late development in a prolonged course of the otitis.

With respect to treatment, Maybaum stresses the following points: *1.* Early myringotomy when infection with pneumococcus type III is suspected and as a means of establishing a diagnosis. *2.* Autogenous vaccine, to be used as soon as the organism is identified. *3.* Complete exenteration of all diseased bony structure, with special investigation of various routes to the petrosa. The recognized surgical procedures should be combined with the administration of sulfapyridine and of pneumococcic serum. *4.* Prolonged observation of the patient after complete healing of the mastoid in order to avoid subsequent and unexpected complications.

Numerous articles have appeared during the past year on the subject of chemotherapy in general, with only incidental reference to its application in otitic sepsis. Such articles, particularly by Long and Bliss[7, 8, 9] and by Powell and Chen,[10] do not naturally render themselves readily condensable for purposes of this review. Applying chemotherapy more specifically to otological problems, Fenton,[11] in discussing its use stresses, first, the importance of adequate surgery without which undue reliance on the drug may prove disappointing; second, the dangers of the drug; third, the masking of symptoms which its use may provoke and which has resulted in unexpected development of complications; fourth, the danger of its indiscriminate use, particularly in the early stages of mild otitic infection; and fifth, the value of blood counts in estimating the postoperative course of any patient being treated with these therapeutic adjuncts. Most of the points stressed in this article crop up in a large number of the various reports of treatment of otitic sepsis by the use of chemotherapy. To this extent, Fenton appears in the rôle of a spokesman for numberless other clinicians whose varied experiences with this newer form of treatment are being now more and more frequently recorded.

McLaurin[12] has an equally good article dealing with sulfanilamide, and appends thereto an excellent and dependable

bibliography. It is interesting to note his forecast of the value of the newer sulfapyridine, a drug not generally available to clinicians in 1939, the lack of which may be responsible for failure in success in the treatment of many pneumococcic infections. Unquestionably, the year 1940 will see a change in this situation with the greater applicability of sulfapyridine to infection due to this organism.

MENINGITIS.

In a review of the literature of 1939, it is at once apparent that a major proportion of the articles dealing with otitic sepsis are concerned with the complication of meningitis. As the whole medical profession now knows, this situation is the result of the amazing therapeutic success in the application to this formerly almost hopeless condition of chemotherapy. In the review of 1938 it was already apparent that such success was becoming evident and the passing of time has more than confirmed this forecast. Numberless individual case reports are now available in both the English and French literature to bear incontrovertible testimony to the value and dependability of this form of treatment in otogenic meningitis. To abstract all such individual reports is utterly beyond the scope of this review, and only those are included in which some striking lesson or unusual circumstance makes the case of particular interest. The majority of such case reports are, however, to be found in the bibliographic references.

One of the most outstanding and comprehensive articles on the treatment of otitic meningitis is that by Cunning.[13] He traces the history of otitic meningitis in the past and quotes Gray's survey of the literature from 1901 to 1935, in which a group of 2,200 collected cases of otitic meningitis showed only 66 recoveries, a mortality of 97 per cent. Other similar series, including one of 101 cases at the Manhattan Eye, Ear and Throat Hospital, from 1926 to 1936, all note the same discouraging situation. The author outlines the treatment and results in 14 consecutive cases of proved otitic meningitis during the past two years. In each case there was a similarity of treatment, including a combination of radical surgical intervention, daily spinal drainage and intensive use of sulfanilamide or one of its related compounds. Such

adjuncts as blood transfusion and intravenous administration of dextrose were employed during the same period. Surgically, the procedure consisted of a simple mastoidectomy with exposure of the dural plate over the temporal lobe, the diameter of removal in the average case being approximately 1½ inches. The wound was temporarily filled with a 70 per cent solution of alcohol, which was allowed to remain in it for about two minutes. The cavity was next dried and the dura split with three or four parallel incisions to permit a free flow of spinal fluid. The external ear was then sutured forward and a doughnut dressing applied in such a manner that with pressure during the bandaging, the cutaneous surface could not adhere to the dural incision in such a way as to interfere with free drainage. Palliative measures consisted in elevation of the foot of the bed to 18 inches, continuous intravenous administration of 10 per cent solution of dextrose by the old fashioned Murphy drip, continued for several days. Transfusions were given as were deemed necessary by the patient's condition, with an average of six in each case, depending on the age of the patient. Daily spinal taps were employed to flush out the meninges, reduce spinal pressure and afford material for examination. Sulfanilamide and neoprontosil were given in extensive doses by mouth, intramuscularly and intraspinally, the dose depending on the age and weight of the patient. Cunning feels it is important to produce a high concentration of the chemotherapeutic agent in the blood as soon as possible and to maintain this as long as necessary. He therefore gives, during the first 48 hours, as large an amount as the patient can reasonably tolerate, after which the dose is cut down as symptoms subside. In several cases the spinal fluid remained free of organisms for several days, after which they again appeared. In the author's opinion the drug should not be stopped altogether for at least one month. Oral administration is usually sufficient, but with patients who are unconscious or who cannot swallow, intramuscular and intraspinal medication must be utilized. The average infection is controlled by a concentration of 8-10 mgm. per 100 cc. of blood but in severe meningeal infection it should be raised to 15 mgm. Of eight patients with streptococcic infection, seven recovered; of five with pneumococcic infection, only three recovered. These patients unfortunately were treated before sulfapyridine was avail-

able. Of the 15 consecutive patients treated, 11 recovered and four died, a mortality of 27 per cent. Cunning feels that sulfanilamide, while obviously extremely important, is apparently not sufficient in itself to produce a cure. On the other hand, surgical intervention in itself is not sufficient and it seems that a combination of the two offers the best hope. It is important in using the drug to start treatment early and that the dose be large and treatment be continued for several weeks after cessation of symptoms. Surgical measures, likewise, should be instituted as soon as the diagnosis has been made. Complete summaries of several of these cases are included in this article.

In a symposium on otitic meningitis, Eggston[14] stresses in comprehensive fashion the bacteriology of suppurative meningitis. He feels that the determination of the specific organism in the spinal fluid is not always as easy as many reports would tend to imply. Organisms vary greatly as to their reaction to the Gram stain, especially the pneumococcus, the influenza bacillus and the streptococcus. The stained smear and the culture frequently do not agree in staining reaction and in morphology. Immediate reliable bacteriological reports are not always easy to obtain, since some organisms are hard to grow and require several days for complete report. There is need for the closest cooperation between the clinical and laboratory staff, particularly at the present time when bacterial therapy is assuming such an important rôle in the treatment of suppurative meningitis. Intelligent sero- and chemotherapy cannot be applied without accurate information from the bacteriological laboratory.

In the same symposium, Dwyer[15] discusses the differential diagnosis between suppurative meningitis, tuberculous meningitis and syphilitic meningitis. If the spinal fluid remains clear on standing for any considerable period of time, one may safely assume that the disease is not a suppurative one. In the presence of syphilitic meningitis, the Kahn and Wassermann tests will be helpful, and in tuberculous meningitis organisms may usually be obtained in a centrifuged specimen or the diagnosis substantiated by inoculation of a guinea pig. Since this disease is not of the fulminating nature of suppurative meningitis, time will usually be sufficient for the execution of these more prolonged tests. Continued turbidity

without organisms suggests an extradural infection for which surgery is obviously indicated. An increased number of lymphocytes would point to tuberculosis or syphilis as the etiological factor. The presence of a large number of polymorphonuclear leukocytes indicates either an extradural infection or a frankly purulent meningitis. Variations in staining reaction of organisms is often a source of perplexity since the same organism may at times be Gram positive or Gram negative, depending on the age and degree of destruction by the infection. Fresh organisms normally showing a positive Gram stain will, if in a degenerate form, change to a Gram negative staining. As exemplified in one case by Dwyer, this may cause confusion between a suspected meningococcic meningitis and a streptococcic form, for which the treatment is entirely different. Cases of otitic origin should, the author feels, in general be treated as streptococcic in origin, regardless of the staining quality of the organism and not as cases of epidemic cerebrospinal meningitis.

Jones[16] feels that a particularly early surgical attack is indicated in otitic meningitis before organisms present gain access to the deeper meningeal structures. Every effort should be made to locate the primary focus, if possible. He discusses the possible avenues of approach as follows: *1.* Preformed avenues of long standing, such as dehiscences, or various natural openings for blood vessels and nerves, especially the facial, and such structures as the ductus endolymphaticus. *2.* Direct destruction of bone as the result of infection. This pathway operates particularly in the presence of an acute infection superimposed upon a chronic phase, affording thereby easy access to the meninges. Infection also may occasionally traverse the path of a previous fracture or may follow dislocation of the stapes incident occasionally to myringotomy.

In discussing the treatment of otitic meningitis, Bowers[17] stresses the importance of early symptoms before actual bacterial invasion of the spinal fluid has occurred. Amongst these are photophobia and other vague eye disturbances, an unexplained elevation of the blood count, actual pain in or behind the eye, unexplained nausea and a vague appearance of hypersensitiveness. He cautions against failure to take into account disease of the sphenoidal sinus, which, as has been shown by Eagleton, is not infrequently the starting

point of a meningitis which may later be looked upon as otitic in origin. Bowers feels that prompt surgery is indicated as soon as the above-mentioned symptoms suggest spinal fluid irritation, even before outstanding changes are evident in the fluid. Too much reliance must not be placed upon sulfanilamide in spite of its obvious usefulness, since later involvement of deep-seated structures may be masked. Suppression of symptoms may not necessarily go hand in hand with absence of pathology. If the drug is likely to prove effective, it will usually do so promptly. Bowers, in company with numerous other authors, is opposed to the routine use of sulfanilamide in cases of uncomplicated otitis media marked by satisfactory recovery without such additional help.

Appelbaum[18] discusses the effect and action of sulfanilamide as it applies particularly to otitic meningitis. He feels that the drug is primarily bacteriostatic, with the additional property of neutralizing bacterial toxins. It penetrates tissue and all body fluids. It is rapidly excreted by the urine and is present in the spinal fluid. The more recently developed neoprontosil, which is now so much in vogue, has only about one-third the amount of sulfanilamide radicle when given in equal doses. It may, however, contain some other therapeutic agent quite independent of sulfanilamide which is beneficial in such infections. Appelbaum feels that the drug is best administered by mouth and is less toxic than is sulfanilamide. The average effective concentration in the blood of sulfanilamide is approximately 10 mgm. per 100 cc., but numerous cases are on record in which from 4 to 6 mgm. appear to have been just as effective and often to be as high a concentration as can be obtained, no matter how much of the drug is administered to the patient. Good results have frequently been reported with as low concentration as 1 to 3 mgm. He sees no essential advantage in intraspinal sulfanilamide but if so desired this drug can be given in a concentration of 0.8 per cent in saline solution. Neoprontosil can likewise be used in this manner. At present neoprontosil is available as a powder to be taken by mouth, a solution to give intramuscularly or intraspinally, and may also be given by gavage. The use of capsules avoids the staining incident to the high solubility of the drug in water. Appelbaum discusses the classical dangers incident to the use of sulfanilamide, particularly anemia, agranulocytosis and dermatitis.

He feels that the customary cyanosis is not due to the formation of methemoglobin but to an unknown oxidation product formed in the blood. Acidosis may be treated by sodium bicarbonate solution or by intravenous sodium lactate. Frequent blood counts are advisable and gastrointestinal irritability may require withdrawal of the drug. Sulfanilamide, according to the author, is of no help in influenzal meningitis (an opinion to which this reviewer takes exception in view of one of the case reports to be included below). This article includes a report of 42 cases of pneumococcic meningitis with seven recoveries but without the advantage of treatment by sulfapyridine.

Pertinent to this line of thought is the report of Falor,[19] who, after recording a mortality percentage of 97 per cent in the Akron Children's Hospital from 1928 to 1937, describes a case of otitic meningitis in which a child, age 6 years, was cured by bilateral mastoidectomy and the use of sulfanilamide. In this case it was only possible to obtain a blood level of 2 mgm. per cent in spite of excessive doses. At one time a lumbar puncture showed 3,100 cells, with the fluid loaded with short- and long-chained streptococci. The blood culture was negative, but the white blood cells rose as high as 42,600. In this case the administration of sulfanilamide was not started until signs of meningitis developed after the operation. It is interesting in this connection to note how many case reports reveal the restriction of sulfanilamide to the treatment of a meningitic complication following aural surgery. There is as yet no uniformity of attitude towards the use of the drug during the stages of simple mastoid disease. It is quite apparent that the drug has an extraordinary ability to control the meningitic complications, providing adequate surgery has been utilized to remove the focus of infection. Whether the meningitis would fail to develop had the drug been utilized earlier is still a matter which time and experience must settle. It is with regard to this earlier administration in the absence of a serious complication that present disagreement and lack of data is most in evidence.

Goodyear[20] has a note of warning in an article on otitic meningitis in which he advises against too early operation on the mastoid cells before some degree of localization has taken place. He feels that such premature surgery may play a part

in spreading infection to vulnerable structures, involvement of which is to be avoided if possible. This controversy has in the past provoked great diversity of opinion and is today far from settled, though there is a tendency on the part of most clinicians to feel that at least in the presence of suspicious meningeal signs early operative interference is indicated. Goodyear stresses the importance of the petrosquamosal suture and the veins running through it as a pathway from the middle ear to the dural covering of the middle fossa, through which infection may readily pass to the meninges. Middle ear infection, he feels, spreads via veins in the submucosa to the intracranial structures rather than via the lymphatics. He is, however, in favor of early operation in the presence of an acute otitis media superimposed upon an old chronic and often cholesteatomatous base. At the outset of signs of labyrinthitis, early operation only tends to spread the infection and some degree of localization should, if possible, be awaited. He stresses the fact, which has so frequently been borne out by the experience of other operators, that meningitis may be a development contemporary with acute otitis media and not a secondary effect spreading from a previous infection in this cavity. In company with other authors, he warns that the sphenoidal sinus must always be suspected in any meningitis following a so-called head cold, even if some suspicion is likewise cast on the middle ear. As a prophylactic measure, he advises thorough operative surgery in chronic otitic infection as a preventive for subsequent meningeal infection.

McCaskey[21] stresses as his main objective in the treatment of otitic meningitis eradication of the etiological focus by either a simple mastoidectomy or a radical mastoidectomy, with or without uncovering the sinus and dura. Labyrinthotomy may be indicated in a secondary revision of any previous operation. In disagreement with some other surgeons, he advises exposure of the dura without opening it. Of almost equal importance is the draining of the cerebrospinal fluid system regularly by lumbar puncture and the use of chemotherapeutic agents with such supportive treatment as the individual case may require.

Something has already been said relative to the possibility, in the use of sulfanilamide, of the masking of important

symptoms which might serve as a valuable guide to the existence of more extensive infection. Bearing closely on this problem is an article by Maybaum, Snyder and Coleman[22] on the value of sulfanilamide in otogenous infection. They mention particularly one patient in whom a meningitis developed two days after the onset of acute otitis media. Large doses of sulfanilamide clinically controlled this infection entirely from the symptomatic standpoint but in spite of this the spinal fluid continued to show evidence of pathology. So soon, however, as a simple mastoidectomy was performed and the focus thus eradicated, the spinal fluid became normal. From this the authors conclude that sulfanilamide frequently acts to hold in abeyance important symptoms and thus possibly to deceive the clinician unless he is fully aware of this possibility. In a second case, acute mastoiditis was treated by mastoidectomy and an operation on the petrous pyramid. The patient recovered but subsequently developed a brain abscess and streptococcic meningitis, the symptoms of which were seriously masked by the administration of sulfanilamide. In the personal experience of the reviewer, this masking of symptomatology by early utilization of sulfanilamide is a serious and important factor in the care of all patients with otitic sepsis.

It seems pertinent now to examine some of the numerous case reports of otitic meningitis treated by chemotherapy, particularly those containing some salient point of interest. The majority of these lack the detailed data of investigative findings which accompany many of the longer articles but, nevertheless, afford valuable information in one's effort to appraise the clinical use of these newer therapeutic measures.

Doane, Blumberg and Teplick[23] report two cases, one of a woman, age 59 years, the other of a child, age 4 years, in which otitic meningitis was treated by mastoidectomy and the administration of sulfanilamide. In each instance the spinal fluid, previously harboring organisms, became sterile in two to three days. In one case the mastoid findings were positive; in the other, negative. The concentration of the drug in the case of the child was only 3.5 mgm. and yet sterilization of the spinal fluid occurred with great rapidity. Both patients received transfusions and other supportive measures. These cases are of interest in view of the wide

age discrepancy, the difference in operative findings and contrasting similarity of clinical outcome. The authors have collected recent reports to the number of 21, with records of 71 cases of otitic meningitis in which the total mortality was only 16 per cent, indicating quite clearly the wonderful progress being made in the treatment of this condition.

Hawthorne[24] records the instance of a child, age 8 years, with symptoms of otitic meningitis, in whom lumbar puncture showed 3,600 cells with 86 per cent polymorphonuclear leukocytes and a smear positive for hemolytic streptococcus. Under the administration of sulfanilamide without any operation on the mastoid, the patient made a rapid recovery. While in this particular instance success was achieved without surgery, it is certainly the opinion of most otologists that it is only with grave risk of subsequent complication that elimination of the primary focus is omitted.

Neal,[25] in summarizing 35 cases of otitic meningitis, records 28 recoveries with 23 operations, all patients being treated with sulfanilamide. She feels that there is no reliable evidence to show that the fatalities were connected either with the use of mastoid surgery or without it. Unquestionably, further experience with this condition and this specific form of treatment will tend to settle the problem of relationship between surgery and the drug.

Yule[26] records the treatment in a case of acute pneumococcic mastoiditis with meningitis. Until within the past few months the great majority of case reports dealing with otitic meningitis have been in instances of infection by the hemolytic streptococcus, for which the drug sulfanilamide is notably appropriate. One encounters in the previous year's literature accounts of fatalities due to the pneumococcus in spite of treatment with sulfanilamide — obviously before the introduction of the newer drug, sulfapyridine. In this particular case, symptoms of meningitis were severe. There were, in addition, suspicions of a lateral sinus infection. After 40 days of illness and terminal 10 days' treatment with sulfapyridine, improvement appeared within the first 12 hours of administration of this newer drug. In all probability, another year's experience will serve to confirm the opinion that sulfapyridine may prove as successful in the treatment of pneumococcic otitic meningitis as has sulfapyridine in the streptococcic form.

In a similar example of pneumococcic meningitis, reported by Cavenagh,[27] a girl, age 17 years, whose spinal fluid showed 6,200 cells with Gram positive organisms, required a radical mastoidectomy in addition to a simple operation, and this in turn was followed by dural decompression and final drainage of the labyrinth through the internal auditory meatus. Gradual improvement and ultimate recovery followed, but with residual facial paralysis. It is quite evident, in this instance at least, that the most radical degree of aural surgery was deemed necessary to effect a cure, but prontosil was the only drug available and simpler measures might have sufficed had sulfapyridine been utilized.

The question of the necessity for surgery as against reliance on sulfanilamide alone in the treatment of otitic meningitis is raised by Hall[28] in his report of a man, age 34 years, with bilateral purulent otitis media, a stiff neck, a spinal fluid containing 320 cells, mostly polymorphonuclears, and the hemolytic streptococcic organism. He was treated by a left simple mastoidectomy and a right radical mastoidectomy and labyrinthectomy. On treatment with sulfanilamide the patient's symptoms immediately abated and for several days he felt clinically well in spite of the continuous presence of organisms in the spinal fluid. Subsequent transfusion resulted in rapid improvement and complete cure except for bilateral deafness. In this instance, extreme radical operative measures were taken in the case of one ear and a more conservative attitude in the other with apparently equally satisfactory results.

Thomas[29] reports a case of otitic meningitis due to what he calls the "minute" hemolytic streptococcus combined with colon bacillus, both organisms being recovered from the spinal fluid. In spite of operation the patient died. This he feels is the first case of meningitis to be observed in which the positive organism was the minute hemolytic streptococcus, first isolated by Long and Bliss in 1934. It is only about one-third the size of the regular streptococcus and its colonies are almost microscopic. Up to 1926 there had been reported only 42 cases of meningitis due to the bacillus coli. Such a mixed infection as was observed in Thomas' case is extraordinarily rare but the authenticity of this dual infection, as manifested by both culture of the blood and that of the spinal fluid, is unquestionable.

Rosenwasser[30] records the interesting story of a girl, age 13 years, with acute mastoiditis, in whom, in order to maintain a sulfanilamide level of 15 mgm., a daily dose of 280 gr. of sulfanilamide was utilized, apparently without untoward effect. She was given supporting transfusions and treated from March until May. Organisms periodically recurred in the spinal fluid in spite of this dosage, together with marked fluctuation in the cell count in the spinal fluid. Ultimate cure resulted. The author stresses the importance of observation of labyrinthine function before operation and feels that normal labyrinthine responses in the presence of meningitis rule out the labyrinth as the pathway of infection.

Philipson[31] had two cases of otitic meningitis, one of labyrinthine origin with organisms in the spinal fluid but with entirely negative operative findings. In the other instance of otitic meningitis with mastoiditis, the spinal fluid showed hemolytic streptococci for 11 days, after which it became negative for a period of time, and then positive again. A petrous operation yielded negative findings, but upon administration of more sulfanilamide a cure resulted. Based on these two patients, with negative operative findings and ultimate cure by means of sulfanilamide, Philipson raises for consideration the above discussed question of the necessity of surgery in the presence of such infection or the reliance on sulfanilamide alone. He likewise points out the value of repeated lumbar puncture in maintaining adequate spinal drainage and feels that this plays a most important part in recovery from such infection.

It has as yet never been claimed that sulfanilamide was particularly effective against infection due to the Staphylococcus aureus hemolyticus. It is, therefore, unusual to read the report of Michels and Conne,[32] who report a case of otitic meningitis due to this organism and treated by sulfanilamide, with recovery. The lumbar puncture showed 620 cells with a positive culture for the staphylococcus. Blood level was maintained at about 15 mgm. per cent, and repeated blood transfusions and intravenous infusions were given. The blood culture was likewise positive for this same organism. Sulfanilamide was given both by mouth and parenterally, and while the patient's response to the drug was not so dramatic as in most instances of hemolytic streptococcic infection, the effect was obvious.

Canuyt[33, 34] records four cases of severe otitic meningitis in which the patient was treated both by surgery and by a modified form of sulfanilamide known in France as Septolix. Theoretically, mouth administration of this drug is sufficient, but in two of the author's patients resort was had to intraspinal injections. He warns against the dangers of the effects of this drug on the patient's sensorium to the point that mental confusion and similar effects may possibly be mistaken for the development of an intracranial complication and thus give rise to inappropriate surgical effort in an effort to deal with it. In this country it has been noted by numerous clinicians that large doses of sulfanilamide may temporarily disturb the patient's mental situation but, so far as this reviewer is aware, the problem has not constituted so serious a difficulty as to cause confusion with respect to actual cerebral pathology. The same author cites a case of pneumococcic otitis in a child treated both by operative procedures and by the administration of sulfapyridine with complete cure. Attempts to secure this result with preliminary sulfanilamide were unsuccessful.

Escat[35] records two cases of otitic meningitis in both of which the patient was treated by surgery and with sulfanilamide. In one instance a cure resulted, the infection being due to the colon bacillus. In the other, with infection due to the pneumococcus, the patient died. In discussing this latter, the author expresses great pessimism as to the treatment of pneumococcic infection with sulfanilamide. In the light of our present knowledge, it seems quite probable that had sulfapyridine been available in this instance, a successful outcome would have been achieved. It is interesting, at least in one instance, to note that infection due to the colon bacillus seemed amenable to sulfanilamide. A further interesting report from France is that of Ombredanné,[36] who described a case of streptococcic meningitis following an acute otitis media, in which the symptoms of the latter were entirely latent at the time the meningitis developed. The latter was cured, apparently in a few days, by the administration of sulfapyridine but at a subsequent date surgery was necessary for eradication of disease in the mastoid process and petrous pyramid. It seems to this reviewer that this is an excellent example of the ability of sulfanilamide to eradicate active infection in the cerebrospinal system without, at the same

time, affecting a definite focus of infection in the mastoid or petrous pyramid. Unquestionably, in this particular situation, had subsequent surgery not been done, meningeal infection would have again recurred as soon as the sterilizing effect of the sulfanilamide had been withdrawn. For this reason it would seem that the greater weight of evidence is in favor of adequate surgery for removal of any focus of infection in spite of the apparent successful effect on the meningeal symptoms of sulfanilamide alone.

A most unusual record is that of Buckman,[37] who reports the recovery from hemolytic streptococcic meningitis, otitic in origin, of a woman at term, who, at the height of her infection, was delivered of a normal living child. Following confirmation of the diagnosis by lumbar puncture, an extensive simple mastoidectomy was done and the drug administered both by mouth and intramuscularly. The patient received seven plain transfusions and two immunotransfusions. At the very height of her infection she went into labor, following which there occurred a profuse postpartum hemorrhage of 600 cc. of blood. Uterine packing was necessary. At birth the baby voided pink urine, indicating the presence of sulfanilamide in his vascular system, but the author unfortunately lost the opportunity to measure the blood level in the infant. Following several days of severe jaundice, the latter appeared to show no ill effects from his or his mother's experience.

Influenzal meningitis has long remained a disease with a high mortality and until recently has only rarely been amenable to any specific serum or other therapy. Long and Bliss have reported that, although sulfanilamide inhibits the growth of this organism *in vitro*, its use in the treatment of clinical meningitis has not been very successful. For this reason, it is interesting to read the report of Brown, Emswiler and Reck,[38] who recently treated a patient with influenzal meningitis following an acute otitis by surgery, prontosil solution, sulfanilamide by mouth and continuous spinal drainage, with ultimate recovery. No specific serum was used. The patient, a child, age 6 years, was operated on by simple mastoidectomy with exposure of a small area of dura which appeared normal. Continuous intraspinal drainage was instituted in the hope of increasing the production of cerebrospinal

fluid and improving the quantity of drainage. On three occasions growth and culture of the spinal fluid showed influenzal bacilli. After three weeks the patient's neurological examination gave entirely normal results, and subsequent convalescence was uneventful. The authors comment on the fact that influenzal meningitis is generally accepted as a primary disease, not associated with any striking infection of the respiratory tract. In this instance symptoms referable to the ear clearly preceded the meningeal signs and typical organisms were cultured both from the mastoid secretion and the spinal fluid. They feel, therefore, that primary mastoiditis was responsible for the influenzal meningitis and that surgical removal of the septic focus was the most important part of successful therapy. It is noteworthy that continuous spinal drainage was successfully carried out on this patient. The author's experience with hypotonic saline solution was not satisfactory, but adequate drainage was maintained with the routine 5 per cent dextrose in saline solution given in the form of a constant, intravenous drip. Numerous other authors, commenting on this form of continuous spinal drainage, feel that it is an unnecessary refinement and the technical difficulty incident to its execution makes it inadvisable. The authors do not definitely claim that either the prontosil solution or the sulfanilamide, which were employed in only moderate doses, were definitely operative in effecting a cure in this instance. They feel, nevertheless, that the drugs were of some benefit and that further study and trial may establish the place of sulfanilamide at least as a partial treatment of influenzal meningitis.

The possibility of a subsequent flare-up after apparent cure in otitic meningitis is well illustrated by a case reported by Teed.[39] In this instance, a patient suffering from left acute otitis media with complicating meningitis was subjected to mastoidectomy and treatment with sulfanilamide. In three weeks' time the patient appeared entirely cured. Three months later, and four days after operation for removal of tonsils and adenoids, there was a recurrence of symptomatic meningitis with all the marked neurological changes attending this disease but without evidence of actual bacteria in the spinal fluid, either on smear or by culture. The tympanic membrane was opened with the release of purulent fluid, and in spite of the fact that this yielded a staphylococcus on cul-

ture, sulfanilamide was again administered, with prompt recovery of the patient. For some unknown reason, the author does not particularly stress the importance of the utilization of sulfanilamide for the original streptococcic meningitis and places greater emphasis on the mastoid surgery. The preponderance of reports of this condition, however, obviously indicate the important rôle played by the drug in numberless instances of such infection.

In stressing the value of the treatment of streptococcic meningitis by sulfanilamide, White[40] reports a case of a boy, age 7 years, suffering from actue otitis media with spontaneous rupture of the drum. Subsequent simple mastoidectomy was necessary with discovery of free pus and extensive destruction of mastoid cells. The dural and spinal plates were apparently normal. On the third postoperative day the patient began to show stiffness of the neck and a doubtful Kernig sign. The mastoid wound was, therefore, reopened and repacked. Meningeal signs continued to develop and spinal examination showed 2,200 cells and a pure culture of hemolytic streptococcus. Sulfanilamide treatment was instituted, together with two tranfusions, and within 24 hours the temperature fell from 104° to normal, never rising to more than 101° subsequently. All neurological signs disappeared within three days. The spinal fluid culture was persistently negative after administration of the drug was begun. On one occasion, tendency to signs of meningeal irritation led to readministration of the sulfanilamide. The patient was discharged, cured, 20 days after operation. The author feels from this experience that sulfanilamide is a powerful ally in combating hemolytic streptococcic infection, that the focus of infection must be eradicated whenever possible, and that the use of sulfanilamide should be discontinued after all signs of meningeal irritation have disappeared only if instructions are given to repeat it on the reappearance of any signs suggestive of return of meningeal pathology. In the absence of free drainage of the focus, a sterile abscess may result, which may be followed by subsequent destruction of bone and serious complicating consequences.

In view of the fact that an otitic meningitis supervened 12 days after a tonsillectomy, the case history reported by Gonne, Michels and Watson[41] is of particular interest. The

frequency with which tonsillectomy is performed and the general freedom from otitic complications makes such a distressing outcome all the more impressive. A girl, age 9 years, had for several years suffered from a chronic otitis media, for which mastoidectomy had been recommended but refused by the parents, who preferred temporizing with tonsillectomy. In the face of typical meningeal signs and a spinal fluid containing 2,500 cells with 90 per cent polymorphonuclears, the patient was operated upon by complete left mastoidectomy. Immediate treatment by sulfanilamide was instituted, with a blood level reaching as high as 20 mgm. per cent, but only after four days were there signs of improvement. Following this, the patient made steady progress and ultimately recovered completely. This case clearly indicates the risk of allowing a definitely chronic, purulent middle ear infection to progress untreated, with the possibility that at any moment, either spontaneously or, as in this case, probably aggravated by the effect of removal of tonsils and adenoids, there may be a sudden flare-up of a serious intracranial complication. Previous consideration has already been given to the question of otitic meningitis due to pneumococcus type III. Extraordinarily high mortality connected with this organism has been repeatedly commented on. For this reason the report by Silverman and Thorner[12] is of more than passing interest. In this instance, a negress, age 47 years, was admitted with obvious signs of central nervous disease and a frank, purulent right otitis media. Spinal fluid examination showed 2,100 cells, of which 95 per cent were polymorphonuclears and a pure culture of pneumococcus type III. It is perhaps surprising that the record indicates the fact that the otological consultant considered this patient too poor a surgical risk for the mastoidectomy which would otherwise have doubtless been performed. On the subsequent day there were 4,000 cells in the spinal fluid, in spite of the fact that the patient had been placed upon adequate sulfanilamide therapy. Four days later, however, and after two blood transfusions, the culture of the spinal fluid was negative and the cell count had fallen to 390. From this time on, continued clinical improvement occurred, with the exception of some right-sided brachial paralysis. The patient was, in addition, a known diabetic, for which treatment was also given. Three months later, after being quite well, the patient suddenly

became ill, with evidence of return of meningeal signs and with 1,640 cells in the spinal fluid. In addition there were signs of definite diabetic acidosis and in spite of treatment for this and the administration of sulfanilamide, the patient died. Here again, is another instance of apparent cure of meningitis, as such, but with neglect of the local focus, although in this case quite justified, there is apparently always risk of recrudescence of a subsequent meningeal infection. Obviously, with this patient the diabetes was a complicating factor, quite apart from the meningitis. While the acute meningeal reaction preceding death from diabetic acidosis might have been occasioned by leakage of necrotic material from the mastoid area, the absence of any organism in the spinal fluid during this terminal episode and lack of postmortem evidence of acute inflammatory meningitis makes reinfection unlikely as a cause of the terminal episode. The authors record the fact that of 130 cases of acute meningitis observed in two years' time, 40 were due to pneumococcus, and 12 to the type III organism. In 10 of these 12 the origin was otitic and the outcome a fatal one in all except the case here reported. Four other cases were treated with sulfanilamide. In one, inadequate dosage was given; in the second, in spite of intensive treatment with sulfanilamide, prontosil and spinal fluid drainage, the patient lived only eight days and died with a spinal fluid too thick to drain. In a third case, meningitis developed a month after mastoidectomy, the patient dying a week after therapy with sulfanilamide. In a fourth case, the patient received sulfanilamide, improved, and was operated on by mastoidectomy, at which an extradural abscess was found. Sulfanilamide was continued and the spinal fluid count reduced, but death occurred from cerebral edema. It must be acknowledged, of course, in reviewing this group of cases that the authors were restricted to the use of sulfanilamide rather than sulfapyridine, with which their results would today be doubtless vastly improved.

PETROSITIS.

From a subject which a few years ago was a definite novelty in otological literature, that of petrositis is now accepted as a relatively well standardized entity with which more and more clinicians are becoming familiar, not only from the standpoint of symptomatology and diagnosis but also from

that of operative and surgical treatment. In discussing the question of what is meant by petrositis, Shea[43] briefly reviews the history of the development of the term and clearly outlines the present recognition of the process of pneumatization, whereby the original diploic bone housing the labyrinth comes to contain cells which at times are liable to extension of infection from the middle ear. In the presence of normal pneumatization, there may be four pathways of infection through the petrous fossa: *1.* superior, from the epitympanic recess above the semicircular canal, extending inward underneath the superior petrosal surface to the tip; *2.* inferior, from the hypotympanum underneath the labyrinth and internal auditory meatus, to the tip; *3.* Eustachian, an anterior route around the Eustachian tube above the carotid artery; and *4.* posterior, beginning at the sinodural angle and extending inwardly in relation to the posterior surface of the petrous bone. Barring a few exceptions, one may assume that the same degree of pneumatization will take place in the petrous pyramids as is found in the mastoid process, the latter serving, thus, as an indicator of the former. Early surgical drainage of the mastoid cells, providing that it is thoroughly undertaken, is the best insurance against extension into the petrous bone. There is a greater tendency for secondary infection superimposed upon a chronic otitis media to extend into the petrous, and the appreciation of this fact is as important in deciding as to the indication for a simple or radical mastoid operation. Shea feels that many failures after radical mastodectomy are due to a chronic petrositis which continues to drain after operation and the discharge from which is blamed upon a patent Eustachian tube. Frequent improvement after a secondary operation for disease inadequately cared for at the primary attack is due to drainage of the petrous routes by uncapping the cells in the epitympanum or the sinodural space. Shea lists as classical symptoms pain in, behind or about the eye of the same side, and pain referred to teeth, particularly in children, often intermittent but always worse at night. Aural discharge is usually constant and only diminished by saturating the patient with sulfanilamide or by rupture of the abscess cavity externally. Fever is not a characteristic finding, though the majority of patients have a slight daily temperature rise, varying sometimes to include a high septic curve.

External rectus muscle paralysis, resulting from pressure on the VIth cranial nerve, is not always a constant finding. Occasionally other cranial nerves may be involved, the VIIth with an exhibition of facial paralysis, the VIIIth with vertigo and nerve deafness, the IXth, Xth and XIth with a jugular foramen syndrome, and the XIIth by atrophy of the tongue on the same side and on protrusion to that side. Shea stresses the great credit due to the field of Roentgenology in the advancement of our knowledge of petrositis. He feels that several exposures must be recorded in different positions intelligently to demonstrate structure and pathology of the petrous pyramid. It is only by Roentgenological study that we may decide that the infection is still limited to an intact petrous bone. This knowledge is important in differentiating the condition which is improving from one which is progressing rapidly toward an inevitable intracranial lesion and possibly death. He lists the following major positions for Roentgenography as follows: *1*. Stenver's position, which he classes as the most useful of all positions and which is used to show the petrous bone perpendicular to its long axis. This is the only position in which the labyrinth can be demonstrated with any degree of consistency and by which suppuration of the apex of the petrous bone can be detected; moreover, tumors of the auditory nerve can be demonstrated by the enlargement which they produce of the internal auditory meatus. Fractures of the petrous bone can be likewise demonstrated which would not show in routine examination. Both sides must be taken for comparison. *2*. The occipital view, or the Chamberlin-Towne position. This position is used to detect erosions of the petrous bone from acoustic tumors and is of value in demonstrating the antral region in sclerotic mastoids. *3*. Anteroposterior view. In this view the petrous portion of the temporal bone is projected through the orbit, giving clear, unobstructed access especially to the inner portion. It is valuable in demonstrating erosion of bone in this locality, tumors of the brain or petrositis from long standing suppuration of the mastoid. *4*. Verticosubmental view. This view is extremely important in the detection of suppuration of the petrous apex.

In the discussion of these four Roentgenological views or positions, full details are given as to the placement of the patient and the position of the head in the actual process of

making the film. Shea feels that the diagnosis of the presence of clinical petrositis demands the surgeon's best efforts. At times a thorough simple mastoid operation will afford sufficient drainage, whereas in other instances radical operation may be necessary. The former is advisable except in the presence of urgent meningeal irritation. In the case with simple mastoidectomy much depends on how recently this has taken place, and temporary disturbing symptoms may disappear upon careful clinical observation. The longer after operation the signs and symptoms of petrositis appear, the greater will be the necessity for further drainage.

A bibliographical list of 40 recent articles on petrositis is offered by Towson,[44] who, in addition, gives a detailed report of a personal case. The patient was a negro boy, age 9 years, admitted with evident symptoms of right mastoiditis. Operation revealed a mixed cellular structure, free pus and some breaking down of the walls and marked softening in the area of the zygoma and posterior angle. A wide area of tegment of the middle fossa was exposed and a large portion of the sigmoid sinus uncovered. Cultures revealed a Streptococcus hemolyticus. Subsequent symptoms quite typical of petrositis developed, and after confirmation by X-ray, a radical mastoidectomy was performed and the semicircular canals and perilabyrinthine structures skeletonized and explored. Some necrosis about the Eustachian tube led to curettage of the peritubal cells, which were found diseased, a finding necessitating opening of a large area along the ascending portion of the carotid artery. The brain was then elevated from the anterosuperior surface of the petrous and a probe passed along this route, but no pus was seen or evacuated. Subsequent recovery was uneventful, except for delayed function of the right external rectus muscle, which, however, subsequently returned. The author raises the question as to whether such an extensive surgical intervention was necessary or not, but feels that investigation of the pericochlear and peritubal areas at least was indicated.

Lillie and Williams[45] present detailed reports of six cases of chronic suppurative lesions of the petrous pyramid in which various pathological lesions of a chronic nature were seen. Two were cases of encapsulated chronic petrositis without apparent otitis media. The conclusion was obvious,

though not comforting, that an apparently healed mastoiditis or petrositis had again become active after a period of five to 10 years. In certain cases there is an obscure clinical syndrome referable to the head, in which the history of an ancient aural disease may assume a place of great importance in determining the causative lesion. Such cases show the futility of attempting to fit clinical material into clean-cut or classical pigeonholes, since the cardinal symptoms and signs of petrositis in the cases here reported were absent in whole or in part. If a serious lesion in the petrous pyramid had not been suspected and an exploratory operation carried out until the development of complete signs and symptoms, the result would doubtless not have been so favorable. The value of exploratory operation in obscure conditions in the temporal bone is obvious. The authors feel that this group of cases demonstrates the necessity of approaching the petrous pyramid without preconceived ideas as regards surgical technique or its adequacy in a particular case for the control of the disease encountered.

It would indeed be carrying coals to Newcastle for this reviewer to attempt to improve on the summary of the literature of 1938 of lesions of the petrous pyramid compiled by Kopetzky.[46] Reference is made, however, to this excellent summary for the benefit of those readers who may wish in investigation of this particular branch of otology to find ready access to the most important work during this period on this subject.

From the standpoint of surgical technique in the treatment of petrositis, Gerling[47] offers a report on the case of a girl, age 12 years, who, after treatment of a right otitis media with success by sulfanilamide and sulfapyridine by mouth, later presented a Gradenigo triad. By means of the Ramadier operation, a huge amount of pus was drained from the petrous apex medial to the carotid artery, together with an extradural abscess. Spinal fluid culture was negative but the cell count was 8,500, with 95 per cent polymorphonuclear leukocytes. Active treatment was undertaken with sulfanilamide and the patient recovered. The author, in discussing the Ramadier technique, cites the difficulty of its execution, the danger of hemorrhage from the internal carotid artery and the possibility of infection of the carotid venous plexus. In addition, the hearing is ruined in most instances.

Reports from the literature indicate that relatively few such radical operations have been undertaken to date and as follows: Ramadier, eight; Barth, one; Mayer, four; Nager, four; Schlander, one; Kopetzky, three; and Lindsay, three. Such surgery indeed demands not only accurate anatomical knowledge but the height of operative courage.

An interesting report is that of Asherson,[48] who discusses the case of a girl, age 9 years, who, after a previous radical mastoidectomy, developed signs and symptoms suggesting orbital abscess or cavernous sinus thrombosis. There was marked edema of both lids and ecchymosis of the conjunctiva. The orbit was explored but no pus found, and external ethmoidectomy yielded equally negative results. The right radical mastoid cavity was then opened, the dura exposed and found congested and bulging, but no extradural abscess was evident and the lateral sinus was normal. After becoming progressively worse for a few days, the eye condition underwent gradual improvement and a complete cure finally resulted, in spite of a right external rectus paralysis which lasted for a short time. The author feels that this unusual complication and possible source of confusion was doubtless due to a petrositis which was cured by simple dural exposure.

Voss[49] raises the question of the possibility of avoidance of the majority of purulent infections of the petrous pyramid. Certain anatomical cell arrangements preclude the possibility of pyramid infection subsequent to middle ear and mastoid infections. In other instances, in which cell arrangement is favorable to the development of infection, prevention is only possible by initial complete anatomical removal of all perilabyrinthine cells. The danger to adjacent structures can be avoided by careful work with proper instruments. Accurate timing of the operation is a matter of experience and inborn judgment, acquired only after repeated contact with these conditions, but in general the author feels that it is better to operate too early than too late. The fact that less complete operative removal of perilabyrinthine infection does not lead to petrositis and the fact that petrositis if symptomatically present gets well without extensive surgery is more a matter of good luck from the arrangement of cells than of good judgment on the part of the operator. Fatal complications undoubtedly often have their origin in a failure

to reach these more inaccessible regions. To substantiate these claims, the author records the fact that he has encountered only three cases of petrous infection in which he himself had done the preliminary operative work on the middle ear and mastoid.

In discussing his methods of procedure in the treatment of petrositis, Jones[50] lays down the following principles in treatment of the wound after operation. The latter is left unsutured and gauze packing is used in such a manner that the maximum amount of space is available for after-care. A small rubber tube or wick of rubber tissue is placed near the discharging sinus, the gauze being packed tightly around the periphery of the wound but loosely around the drainage. The essential object is to keep a small pathway clear through which pus is draining, since granulations tend to form quickly and obstruct this. Such granulations may for weeks require frequent removal with a ring or small Spratt curette, with supplementary use of a small suction tube to promote drainage. With disappearance and cessation of all discharge, a secondary plastic procedure produces good cosmetic results, and in the case of a radical mastoid operation the canal flap may be made at the time of this plastic procedure. Jones feels that skin grafts are contraindicated because of the possibility of the epithelium sealing off a partially resolved infection. In postoperative treatment following approach by the Gasserian ganglion route, Jones stresses the precaution of transfixing any drain to the outside of the skin lest it be lost or prove difficult to recover. Also, one must guard against drains adhering to the dressing lest they inadvertently be pulled out and prove difficult of replacement.

Bateman[51] records two cases of apical petrositis, one in a female, age 28 years, the other in a boy, age 14 years. The author lays stress on the type of pain in apical petrositis, which, although on the affected side, is often very difficult to localize, and, although sometimes behind the eye, is on other occasions not recognized or admitted to be such by the patient, who may sometimes be found crying in bed because of this discomfort. Pain attributable to irritation of branches of the Vth nerve is more definite and peripheral and often affords an easier point in diagnosis. Persistent discharge may be the result either of ordinary groups of cells which have not been

adequately removed, or from infected petrous cells. In Bateman's cases the temperature was not high and X-ray was not conclusive in either instance. Oftentimes normal skulls may show suspicious increased density in the region of the petrous pyramids and one cannot place undue reliance on this diagnotic aid. On the other hand, the absence of positive X-ray findings does not rule out pathological bone absorption. Diagnosis is admittedly difficult, but is made on persistent discharge after complete operation and vague retroocular pain, X-ray findings and Vth or VIth nerve involvement. Treatment should be operative. One may either adopt a fixed type of operation in the manner of Ramadier or Lempert or Eagleton, or one may search for a track of infected cells leading to particular places, according to the symptoms at hand.

LABYRINTHITIS.

As was the case in last year's review, the paucity of reports concerning infection of the labyrinth attests the fact that this condition is still the rarest of the otitic complications. For this reason it is the more surprising that from any one institution there comes an account of experience with work on the labyrinth in 244 cases collected between 1923 and 1938 by Frenckner[52] at the Sabbatsberg Hospital. Among these there were 91 instances of labyrinthitis circumscripta, 91 of diffuse serous labyrinthitis, 55 of manifest purulent infection, and seven of latent purulent infection. In the circumscript form of labyrinthitis, operation is indicated by meningeal irritation or extraordinarily severe subjective or objective syptoms. In diffuse serous labyrinthitis, if accompanied by marked reduction in cochlear or vestibular function, the requirements from an operative standpoint are the same as for purulent labyrinthitis. In acute diffuse suppurative labyrinthitis, some form of labyrinth operation should be performed for meningeal irritation or any other form of intracranial complication and opening suppurative fistula or evidence of sequestration. In Frenckner's series of 60 labyrinthectomies, obviously an amazingly large group for any single institution in 15 years, there were 18 in which the signs of meningeal irritation were very slight. There were no postoperative complications and all patients recovered. In 39 cases definitely combined with meningitis at the time of operation, 14 patients, or 36 per cent, died of intracranial

complications. This article comprises a most detailed study of this clinical condition and merits study in the original publication.

In view of the present vogue for chemotherapy in the treatment of all forms of otitic infection, it is not surprising to read the report of Layton,[53] who raises the question of lessened frequency for the necessity of radical labyrinthine surgery in the presence of possibility of treatment by sulfanilamide. He suggests a thorough trial of the drug first with reservation of operation until later necessity for its use is proved. This attitude is quite in keeping with that expressed by numerous other authors in a general consideration of otitic complications, and may possibly lead to still further decrease in the number of labyrinthine complications requiring radical surgery. Further, it would seem that the high vascularity of the membranous labyrinthine structures would render them ideally susceptible to treatment by chemotherapy, and the greater vogue for more timely and adequate mastoid and petrous surgery will undoubtedly continue to decrease the number of cases of labyrinthine infection which are the result of the spread of contiguous bone disease to the labyrinth itself.

SINUS THROMBOSIS.

In an article commemorating the fiftieth anniversary of the first successful operation for thrombosis of the lateral sinus, Cody[54] presents the findings in a series of 518 mastoidectomies on private patients, among whom there occurred 16 instances of thrombosis of the lateral sinus, a percentage of three. In 11 instances this condition was a postoperative complication in acute mastoiditis, and in five of chronic mastoiditis. Fairly uniform treatment in this group consisted of operation on the sinus with ligation of the jugular vein, except for occasional resection, when the vein was thrombosed. The sigmoid sinus was regularly uncovered first because of the possibility of periphlebitis or perisinus abscess, and a small incision made through the wall of the sinus. In 14 of the 16 cases a thrombus was present. The internal jugular vein was usually ligated below the tied facial vein and cut between the ligatures. The sinus is then exposed distally by bone removal until free bleeding occurs, and proxi-

mally until either free bleeding appears or the course of the sinus toward the jugular bulb is straight, and the proximal thrombus toward the bulb is removed by suction. Bleeding is controlled by rolls of iodoform gauze, the size of a pencil, packed between the cranium and the sinus. In the absence of bleeding from the proximal end of the sinus, the latter is left open and irrigated daily. Packs are removed on the fifth day, usually with the aid of a general anesthetic. Four of the 16 patients afflicted with sinus thrombosis died, a mortality of 25 per cent. One of these was admitted to the hospital with advanced meningitis and died within 48 hours, this case, therefore, being omitted from statistical consideration. A necrotic sinus wall was demonstrated in five patients, of whom two died. No thrombus was found in two patients, of whom one died. A thrombus was present in 13 patients, three adults, of whom one died, and 10 children, of whom one died. Analysis showed a manifest thrombosis in seven cases, a latent one in five, and a postoperative one in three. A sinus operation done at the same time as the mastoidectomy is recorded in seven patients, of whom two died, and after the mastoidectomy in eight, of whom one died. Cody stresses the value of blood transfusion as a therapeutic agent; first, to remedy a secondary anemia from the sepsis; second, to provide sufficient antibodies to affect the virulence of the invading organism; and third, to increase the amount of available antibodies, either by immunizing the donor or by increasing the number of transfusions. Though sulfanilamide was used in only two cases, the author feels that its use presents possibilities of a brilliant future and that its use will continue to prove more and more valuable with added experience with this particular form of complication of mastoid infection.

The therapeutic results in 30 cases of otogenic sepsis of thrombotic origin are recorded by Joannovich.[55] Eighteen of the cases followed acute purulent middle ear inflammation, and 12 followed exacerbations of a chronic purulent otitis media. In 21 instances the attending organism was the streptococcus, in seven the pneumococcus, and in two cases the enterococcus. In 28 instances, exposure of the lateral sinus was accompanied by jugular ligation, with 18 recoveries and 10 fatalities. In two cases the vein was exposed without ligation, with a 50 per cent mortality. Thus, among the 30 patients there were 19 recoveries and 11 deaths. In the 12

instances of streptococcic septicemia in which simultaneous treatment by the use of Vincent's streptococcic serum was adopted, there were 11 recoveries, or 91 per cent, and one death, in spite of the fact that these were among the most serious instances of the disease. In contrast to this, there were nine other cases of streptococcic septicemia in which Vincent's serum was not employed, with only four cures, or 44 per cent. This result leads the author to the conclusion that this serum was of paramount importance in the recovery of these patients. There is no mention of the use of chemotherapy, either as a supportive measure or in conjunction with operative and serum therapy, so that one is left uncertain as to whether this adjunct now so widely used in this country might possibly have bettered the author's statistics.

In describing his personal preferences in postoperative care after surgery in the sigmoid sinus, Jones[56] recommends procedure on the sigmoid first and, after changing to an entirely new, sterile setup, the operation on the jugular vein. He advocates leaving the mastoid cavity completely open with the exception of a stay suture of silkworm gut. Iodoform gauze tampons are placed over each end of the exposed sinus, the remainder of the cavity being packed with the same material. The neck wound is closed with Michel clips, though one may also use a subcutaneous suture of No. 1 catgut with excellent cosmetic results. A small cigarette drain is placed in the neck sutures parallel to the incision, with its exit at the lower extremity of the wound. An oiled silk apron is fastened to the neck by collodion, being turned up over the mastoid dressing during the neck dressing and after the completion of the latter, turned down as a cover and protection against any possible seepage from the mastoid wound. With favorable progress, the sinus dressing is not disturbed until the fourth or fifth day, at which time the gauze packing is thoroughly soaked with peroxide. By careful removal of the gauze and frequent applications of peroxide, the first part of the dressing can be finished without disturbance of the wound or discomfort to the patient. With children or very nervous patients, the first dressing may be done under anesthesia. In the removal of the plugs occluding the sinus, the lower plug is removed first. Peroxide is again applied and the plug firmly grasped with mouse-tooth forceps. Gentle side to side and rotary traction is usually effective. Providing bleeding

occurs, the plug is left in place and removal again tried at daily intervals until accomplished. The upper plug is handled in the same manner. With the plugs successfully removed, secondary closure of the wound is carried out, with treatment the same as in any complete mastoid operation.

Ersner[57] stresses the danger of assuming that any signs of sepsis in a patient suffering from otitic infection necessarily indicate an infection of the lateral sinus. Abnormal temperatures, particularly of the septic or steeplechase type, chills, sweating and convulsions may all be present without necessarily indicating the presence of lateral sinus sepsis. Erysipelas, pneumonia, meningitis, tonsillitis, infection of the opposite ear, pyelitis or cervical adenitis may at one time or another produce one or more of these complicating symptoms. The distinct clinical and pathological entity of acute hemorrhagic mastoiditis will not infrequently give such typical signs of sepsis, prostration, chills, positive blood cultures and occasionally a choked disc, as to lead one to suspect phlebitis or thrombosis of the lateral sinus, whereas in actuality these symptoms are due to hemorrhagic mastoiditis. Even unduly early operation on the mastoid process, before localization of the infection has taken place, may lead to an ensuing stormy course, with protracted convalescence and possibly with septic symptoms closely simulating sinus phlebitis or thrombosis. The author feels that there is no emergency requiring immediate institution of surgical procedures in phlebitis and thrombosis of the lateral sinus, and that conservative measures may reveal the sepsis to be due to other causes. Such symptoms frequently lead to the conclusion that phlebitis and thrombosis of the lateral sinus are responsible for them in almost 100 per cent of the cases. In actuality, the incidence of sinus thrombosis and phlebitis in otological disease is only 2 per cent. Each case of sepsis, therefore, is entitled to deliberate and thorough consideration from both medical and surgical standpoints before erroneous conclusions are drawn as to the condition of the lateral sinus.

In discussing the value of blood cultures in otitic sepsis, Goldman[58] believes that early diagnosis of lateral sinus thrombosis before a manifest clinical picture has appeared is of great importance if operation is to diminish morbidity, metastases and possibly mortality. He feels that adequate and

complete studies of the blood culture will go far to help in achieving this end. Bacteremia is the most constant feature in cases of thrombosis of the lateral sinus, the micro-organism usually isolated from the blood stream being the Streptococcus hemolyticus. In over 95 per cent of the cases of proved bacteremia associated with otitic sepsis, this organism has been found. Success in demonstrating its presence depends to a large degree on the method used in taking and cultivating the blood. The interpretation of blood cultures requires mature laboratory experience and the methods and media utilized are of great importance. Goldman feels that once even a small number of hemolytic streptococci are found, the diagnosis of lateral sinus thrombosis or thrombosis of the jugular bulb is established, provided all other clinical possibilities have been excluded. He feels that the utilization and interpretation of blood cultures is also of great value in the postoperative care of the patient.

A most comprehensive report on the subject of thrombosis of the lateral sinus is that of Druss,[59] who offers a detailed study of 15 cases of this condition studied at postmortem, with histological examination of the tissues. Sixty-two patients with sinus thrombosis were treated at the Mt. Sinai Hospital from 1928 to 1935. Of these, 44, or 71 per cent, recovered, and 18, or 29 per cent, died. It is from these 18 fatalities that the author makes his present study. Associated complications revealed by postmortem examination in these 18 cases were 12 instances of meningitis, seven of petrositis, four of labyrinthitis, two of abscess of the brain, one of subdural abscess, and one of cavernous sinus thrombosis. Meningitis, the most dreaded complication, is felt by the author to arise either by extension through the blood stream, by retrograde extension of the infection in the multiple tributaries traversing the subarachnoid and subdural spaces, and by direct extension to the inner wall of the sinus. The author feels that the jugular bulb is frequently the seat of involvement and it was found to be diseased in each of the 15 cases studied, its infection being usually secondary to infection from the sigmoid sinus. More rarely, it takes place directly from the tympanum, in which case it is known as primary thrombosis of the bulb. In surgical treatment of thrombosis, Druss advocates the classic operation as recommended in 1884 by Zaufal, which includes ligation of the internal jugular vein, incision

and removal of infection in the sinus, and obliteration of the latter in the customary manner by packing.

A clinical report of a case of great rarity is that of Smith,[60] who describes thrombophlebitis of the lateral sinuses complicating bilateral acute mastoiditis. The literature to date records three cases of bilateral thrombophlebitis of otitic origin with recovery following ligation of both internal jugular veins and obliteration of the sinuses. There have been recorded also six cases of bilateral thrombophlebitis of similar origin with recovery following ligation of one internal jugular vein and obliteration of both lateral sinuses. In practically all of these cases, bilateral ligation was not simultaneous and an interval of at least a few days intervened between the two procedures. It is the author's opinion that this condition occurs more frequently than is customarily thought to be the case and that the small number of reports is due to the belief that a fatality would ensue if both main venous channels were obliterated. The cases already reported seem to establish the fact that this is not true providing sufficient time elapses between the operations. The author adds a detailed experience of his own with bilateral thrombophlebitis with blood stream infection due to the hemolytic streptococcus. and with recovery after operation.

The Bacillus proteus is an infrequent causative organism in otogenic infection and is associated with an extremely high mortality. Since 1912, only nine cases have been reported in the literature, seven patients giving a history of chronic otitis media. All of these were operated upon, and four had the lateral sinus obliterated and internal jugular vein ligated. The mortality was 89 per cent, three patients dying of meningitis, and five of overwhelming bacteremia. After summarizing the literature to date, Gerzog[61] adds a personal case. A girl, age 12 years, had had a simple mastoidectomy at the age of 8 years. Signs and symptoms developed indicating a second or recurrent mastoiditis and thrombosis of the lateral sinus. Immediate operation was undertaken and the sinus found inflamed, discolored and covered with granulations; on opening its wall the lumen was found to be filled with foul-smelling pus. The jugular vein was also ligated. The bacteriological report of a pure growth of Bacillus proteus was returned and immediately a stock bacteriophage was given

intravenously for the next seven days. After 10 days, improvement appeared and the blood culture became negative, although the lateral sinus still drained pus, and for this reason justified continuation of the administration of the bacteriophage for four more days. This rare organism was found not only in the pus from the mastoid and lateral sinus but also on culture of the blood. The author feels that the bacteriophage was the most important essential in the cure of the patient.

The point of view of a trained bacteriologist should be of interest in consideration of the immunological aspects in sinus thrombosis. This subject is interestingly discussed by Schwartzman,[62] who recounts his experiences with blood invasion by the hemolytic streptococcus, particularly in sinus thrombosis. He feels that the stages of the development of this infection are as follows: invasion of the thrombus by bacteria, multiplication of the bacteria in the thrombus and, finally, either destruction of bacteria in the thrombus by formation of the fibrin and thrombus organization, or survival of bacteria, with destruction of the thrombus and dissemination of the infected thrombotic particles by means of the circulation. The micro-organisms possessing the power to dissolve fibrin and thus prevent organization of the sinus are those having the best chance to disseminate the infection and invade the blood stream. The Streptococcus hemolyticus is the only micro-organism possessing strong and specific fibrolytic agents and hence is practically the only micro-organism invading the lateral sinus after mastoiditis. The author fails to see the rationale of jugular ligation in the treatment of this condition. He feels that this operation is analagous to closing up any other infectious focus instead of allowing it to drain. Inflammatory reactions of the remaining parts of the infected vein are, he feels, never able to sterilize the infected focus, some organisms always remaining at the site after operation, even following vein excision. He acknowledges, however, the possibility that ligation may limit the process and prevent secondary thrombi, which may be more harmful than the primary thrombosis. In addition, the fibrin eventually becomes immune to the dissolving effect of the streptococcus and may then help to destroy the bacterial invader through organization of the primary thrombus itself. The author feels that there is still a wide opportunity for bacteriologists to inves-

tigate the immunological aspect of sinus thrombosis as an aid to the otologists who have so demonstrably done their part from a surgical and pathological point of view.

BRAIN ABSCESS.

In discussing the surgical management of otogenous abscess of the brain, Adelstein[63] feels that abscess of the brain is not a surgical emergency and that the early, extremely high mortality was due to the practice of surgical intervention as soon as the diagnosis of abscess was made. Among 19 patients mentioned by Gorham, there was not a single recovery after operation before encapsulation was complete, and within six weeks after initial cerebral involvement. Operation before complete encapsulation, according to the author, is distinctly dangerous and rarely is early intervention necessary. The various techniques of surgical drainage are described in some detail, including those of Macewen, Mosher, King, Hart and Adson, and Craig. More representative of the conservative attitude are the techniques of Dandy, and Coleman and Grant, all of whom have had amazingly successful results and a far higher percentage of recovery than formerly appeared possible. The author's own technique has been uniformly conservative. The abscess is first localized by careful neurological study, encapsulation is always awaited, the patient receiving supportive treatment during the encephalitic stage for at least four weeks after initial cerebral involvement. A small trephine opening is centered as closely as possible over the site of the abscess and its bony margin carefully sealed with bone wax to close off the diploic spaces and prevent contamination. A nick is then made in the dura large enough to allow an exploring cannula to pass into the abscess and drain it. For drainage in the majority of cases, the author uses a one-eye, soft rubber catheter of about size No. 16. The abscess capsule is discovered by careful needling with a blunt brain cannula. Pus is not allowed to gush out and contaminate the field, but restrained by keeping the stylet in place. A rubber catheter is measured off to a correct length and inserted into the cavity of the abscess as gently as possible. With the drain in place, the content of the abscess is allowed to flow gently into an aspirator with little or no contamination of the wound. The catheter is then carefully sutured to the edges of the dural incision, the fascia and the cutaneous edges of the

wound. Care is taken that the sutures in the drain do not enter its lumen, contamination of the edges of the wound and subarachnoid space being thus averted. A safety pin still further prevents dislodgment of the drain, with firm taping to the adjacent scalp. In this manner, no drain has been lost or dislodged. The restraint of the immediate flow of the contents of the abscess avoids collapse of the walls and any tendency to multiloculation. So long as the approach to the dura is kept clean, the damage is minimal, even if no abscess is located. If contamination of a clean brain is induced by exploration, the damage may be irreparable. The wound is dressed daily and the drain shortened as the healing process causes the catheter to extrude. No washing or irrigation is carried out and the drain is not manipulated under any circumstances except apparent obstruction. The drainage period may last as long as six weeks to three months. The author reports three personal cases of otogenous abscess of the brain, two in the temporal lobe and one in the cerebellum. In all three instances the abscesses were secondary to otitis media and mastoiditis, and in all three a cure resulted. He feels that in all surgeons' hands the highest percentage of recoveries will be obtained by drainage by simple methods, by which excessive trauma and spread of the infection are avoided.

Analyzing 27 cases of histologically verified abscess of the brain, Alpers[64] attempts to answer the following questions: *1.* How does encapsulation take place? *2.* When can it be said to have occurred? *3.* What relation does it have to the bacterial content of the abscess? *4.* What should be its relation to operation? Careful histological study of abscesses is made, with special attention to the necrotic zone or cavity of the abscess, the reactive zone adjacent to this, the fibrous zone constituting the most important feature of development of the capsule, and the encephalitic zone or reactionary area within the brain tissue itself. The author feels that the formation of the capsule is dependent on the following variable factors: *1.* the nature of the invading organism, some organisms being more prone to produce capsule formation than others; *2.* resistance of the host, an intangible factor not easily measured and difficult to evaluate. In general, the greater the resistance of the host, the more definite the possibility of capsule formation. From the clinical standpoint, Alpers feels that the longer one waits before operation, the

better is the chance of encountering a walled-off abscess, but it is well known that sometimes no encapsulation has occurred after several months. In most instances, if the situation is not too acute, it is advisable to wait three weeks, and preferably four, before undertaking operation, if possible, except in desperate situations which require immediate intervention. If only encephalitis is present and no abscess is formed, intervention will do nothing more than satisfy anxious relatives. It may be advisable, first, to remove a possible focus of infection in the sinuses or mastoids and then adopt a period of watchful waiting in the hope of subsequent encapsulation. Encapsulation does not necessarily mean inactivity within an abscess, for virulent bacteria may, if permitted to escape, produce a fatal meningitis. Only occasionally is a sterile abscess encountered. Active encephalitis may surround an encapsulated abscess and knowledge of its presence is important, but as yet no signs have been found which may be regarded as characteristic of this encephalitic reaction.

Kahn[65] describes the technique for the injection of colloidal thorium dioxide into the cavity of a brain abscess in order to render it visible Roentgenologically. A previous trephine is made through which to explore the abscess and for purposes of drainage. If capsule resistance is felt, a dull cannula is plunged in. If the capsule is not firm, further delay is advisable before proceeding with tapping. If pus is evacuated, about 6 cc. of the solution is then introduced, with the following advantages: *1.* The abscess is visualized directly. This enables it to be attacked at its most superficial point. *2.* The colloidal thorium dioxide particles are phagocytosed by the cellular elements of the capsule so that increase in the size of the abscess is demonstrable, even though pus and the contrast media do not mix freely. *3.* It minimizes the risk of the capsule becoming lost in the depth of the brain substance at the time of drainage. *4.* The method is valuable if the Vincent technique of excision of the abscess by open operation is employed. *5.* It facilitates fluoroscopic control when the abscess is treated by Dandy's method of repeated tapping. This method clearly shows a tendency for deeply seated abscesses to migrate towards the surface, with spontaneous extrusion or surgical excision.

Krainz[66] discusses the disagreement as to the treatment of brain abscess resulting in a general recovery percentage of

only 30 to 35. His personal experience since 1927 has included 10 temporosphenoidal and one cerebellar abscess. Of the former, nine were on the left and one on the right, six patients were cured and four died. The abscess followed acute otitis media in four instances, and chronic otitis with cholesteatoma in six. Of the acute infections, there were three instances of cure and one death; of the chronic, three instances of cure and three deaths. The one instance of cerebellar abscess occurred after cholesteatoma and resulted in a cure. The author feels that the prognosis is better with chronic otitis media as an etilogic factor because of the greater tendency to capsule formation, whereas in acute infection the tendency is more towards an unlocalized encephalitis. It is hard to tell whether a capsule is present or not, even by speculum examination. Krainz feels that more important than the operative technique is the after-management. He believes in a wide opening, thorough removal of pus, irrigation with peroxide and packing of the cavity with iodoform gauze, keeping the opening in the dura apart with a wide, short, brain drain. Prolonged after-care is important for success. The shortest time of healing of the cavity in his cases was six weeks, and in most instances ran on to three months. It is important, he feels, to keep the walls of the cavity apart until the latter fills in with granulation tissue and that pocketing must be prevented. To this end, he feels that packing of the cavity is important. The latter should be changed daily, at least during the major part of the convalescent period. Iodoform tends to stimulated granulation formation. Aeration of the cavity is important to prevent formation of small accessory pockets and to reach any smaller adjacent accessory abscesses which may be present. In all this postoperative care, great gentleness is necessary in its execution. The author is opposed to prolonged packing left in the abscess cavity without repeated change. Frequent bacterial control is necessary for the detection of any unopened accessory abscesses. From this reviewer's point of view, it must be asserted with respect to the above that this technique of repeated packing would hardly meet with the approval of the majority of American surgeons dealing with brain abscess, most of whom stress the minimum amount of postoperative manipulation possible.

In describing his technique for the handling of a brain abscess, Kulenkampff[67] advocates wide decortication with dia-

thermy, removal of the roof and the exclusion of all foreign bodies from the cavity, barring possibly gentle irrigation of pus if this is unduly thick. The wound edges are sewn back to keep the approach open. Operation is undertaken under local anesthesia, supplemented with a superficial inhalation anesthesia. The author feels that cerebellar abscesses are less favorable because harder to find, harder to diagnose and harder to drain, and that diagnosis is easiest in left-sided temporosphenoidal abscess because of obvious speech disturbances. The patient, however, may not complain of this difficulty until it is called to his attention. The fundus examination may show changes on the affected side and sometimes a fixed pupil will be present. The presence or absence of fever is of little diagnostic help, and the evidence of early meningitis usually indicates a hopeless situation, with a break into a ventricle. Lumbar punctures may help but should be avoided in a suspected cerebellar abscess. Rare recovery may occur after a break into the ventricle if the latter walls itself off and drains externally. The author pleads for early mastoidectomy and thorough exenteration of all cellular leads to avoid cerebral complications.

Sjoeberg[68] reports a case of a man, age 33 years, with chronic otitis media and cholesteatoma, who suddenly became unconscious, with meningitis due to perforation. Operation revealed an extradural and cerebellar abscess, for which drainage was instituted, together with the intraspinal and intravenous administration of prontosil. The meningitis subsided in five or six days and the patient was ultimately cured. This is one of the few examples of the use of chemotherapy in the treatment of meningitis secondary to a brain abscess. In addition, the author summarizes his experience with otogenous brain abscess at the Sabbatsberg Hospital. There were twice as many abscesses in the temporal lobe as in the cerebellum. The mortality after acute otitis was 76 per cent as compared with that after chronic otitis, which was 51 per cent; the total for all abscesses being 66 per cent. In general, the prognosis was better in cerebellar than in temporal lobe abscess. In company with many other American surgeons, he feels that surgical therapy should await encapsulation, which in general does not occur until at least three weeks after onset of symptoms. The degree of brain pressure and not the amount of infection is the major criterion and is

determined by the presence of slow pulse, fundus changes, field examination and general neurological findings. Lumbar puncture before drainage is risky, particularly in the presence of cerebellar abscess. Surgical drainage is affected by a longitudinal incision in the dura, made by diathermy and followed by puncture, with slow aspiration. Following this, a wide excision of the wall of the abscess is carried out by diathermy, with inspection of the lining of the cavity by means of the nasal speculum. A coarse rubber drain is inserted and sewn to the skin and left *in situ* for five to six weeks. The author advocates sulfanilamide by mouth as a preventive of a possible complicating meningitis, at times supplemented by intramuscular and intraspinal administration for not more than one week, watching for signs of intoxication from the drug.

Laskiewicz[69] contributes an extremely valuable and detailed monograph occupying a supplementary number of the *Acta Oto-Laryngologica*, on the subject of brain abscess of otitic and nasal origin. This monograph is too lengthy for inclusion in the present review but is a most comprehensive study of the subject and merits reading in the original French by anyone wishing to pursue this subject further.

In discussing after-treatment in brain abscess of otitic origin, Tobey[70] feels that the principal factors to be taken into consideration are: *1.* the time of drainage; *2.* the site of drainage; and *3.* the method of drainage. With respect to the time, he believes that, ideally, one should await encapsulation, which usually occurs during the fourth or fifth week, or sometimes much earlier; however, in an acute stage with high fever, prostration and increased leukocyte count in the spinal fluid and signs of a beginning meningitis, earlier intervention may be necessary. In choosing a site for drainage, one must presuppose that a thorough mastoidectomy, either simple or radical, has already been done. In the presence of evidence of intracranial involvement, a thorough exposure of the dura in the middle and posterior fossae is a necessity at the primary operation. Evidence of a direct extension of infection from the mastoid process or middle ear is not usually found in acute brain abscess, but in subacute or chronic abscess there is usually evidence of either caries of the tegmen with granulation upon a thickened dura, or occasionally a distinct

fistulous tract connecting with the abscess. Under the latter circumstances, otologists customarily institute drainage in this area. On the other hand, in acute abscess and in chronic abscess developing after healing of mastoid or middle ear suppuration, the point of election is through a clean area as nearly as possible directly over the site of the abscess. The author then describes the most acceptable form of drainage, varying from simple tapping to decortication, and the use of the Mosher wire basket drain. In this latter procedure, the use of the diathermy knife has been particularly helpful. Whatever the method used, maintenance of adequate drainage is essential in postoperative care. In the use of the wire basket, daily curettage of the inner surface is necessary to remove granulations which spring up and project through the wire mesh, interfering thus with drainage, as well as with inspection of the cavity. After an average period of four weeks for drainage, the rubber tube, which has been gradually shortened, may be removed, or the wire basket drain either spontaneously extruded or deliberately removed. Irrigations must be used with caution and with provision for adequate return flow. Daily dressings should be done, preferably by the surgeon himself. The essential complication most to be feared is a spreading encephalitis, which may terminate in septic meningitis. With possible return of intracranial pressure signs, re-exploration may be necessary.

Tempea[71] describes the case of a man, age 30 years, who following a chronic otorrhea of five years' duration, showed typical symptoms of otitic meningitis. For this a Neumann operation was carried out with apparent success and with abatement of meningeal signs. In 10 days, however, a rising leukocyte count was noted and a definite slowing of the pulse, with vomiting and hiccoughs. With the patient in a state of coma, the wound was reopened and the temporal lobe packed. At a depth of 1 cm., 250 gm. of pus were withdrawn, and further amounts were recovered by moving the patient's head about in different directions. Convalescence was in the main satisfactory with the exception of some fluctuations in temperature, which were at one time thought to be due to malaria and which were treated with quinine in spite of the persistent failure to recover the malarial parasite. The final successful outcome in this case was, the author believes, due, first of all, to the suspicion of the possibility of brain abscess in spite of

the minimal symptoms; second, to the complete removal of pus at the original aspiration; third, to maintenance of drainage through a tube; and, finally, to the accessory development of a fixation abscess. No form of antibacterial, intraspinal or intramuscular serum was used in this case.

Van den Wildenberg[72] reports the case of a cerebellar abscess of otogenic origin in a child, age 6 years, successfully operated upon. The author stresses the seriousness of cerebellar abscess, attributing this, on the one hand, to the possibility of encephalitis, difficulty of drainage and the tendency to relapses; and, on the other hand, the possible coincidental development of other otogenic complications. He notes that the major large series of statistics show a percentage recovery in cerebellar abscess of about 15, and in cerebral abscess of about 35. The patient was thought to have an abscess of the cerebellum, the diagnosis based largely on the presence of bilateral choked disc, a right external rectus paralysis, loss of weight, cerebral vomiting, nystagmus and anesthesia of the right cornea. Spinal fluid examination was negative. Ataxia was marked and an examination of the ear revealed nothing abnormal from the point of view either of hearing or vestibular function; however, on the strength of a previous suspected otitic infection, a right-sided mastoid operation exposed the cerebellum and laid bare the sigmoidal sinus. Hemorrhage from the mastoid vein prevented further interference, but no pus was observed and anatomical conditions appeared normal. During the following day, all of the symptoms of cerebellar abscess became more severe, together with diplopia, vomiting, opisthotonus, repeated yawning and incontinence of urine and feces. Subsequently, a needle was introduced at Trautman's triangle and a teaspoonful of pus was withdrawn, examination of which showed no microorganisms. After nine weeks, drainage from the abscess ceased and symptoms disappeared except for slight atrophy of the right disc. This case is unusual because of its obscure etiology, there being very little evidence of either acute or chronic otitis. The ataxia and nystagmus were important leading signs, indicative of cerebellar involvement.

Audry[73] makes a distinction between cerebellar abscesses of anterior and of posterior variety. The anterior variety is situated anterior to the lateral sinus, while the posterior form

is on a level with or behind the lateral sinus. A syndrome of symptoms usually accompanies the anterior variety, which is essentially labyrinthine in nature and exhibits labyrinthine deafness and lack of response to the caloric test. There is, moreover, a spontaneous nystagmus and, if the patient is in a state of coma, a conjugate deviation of the eyes. With the posterior form of abscess, these labyrinthine signs are lacking beyond a slight nystagmus when looking to the extreme lateral position. Reaction to the caloric test is either normal or exhibits a vestibular hyperexcitability. The author records the case of a young patient who, since childhood, had been subject to painful attacks of middle ear infection. Since February, 1938, the ear had drained continuously. About the first of January, 1939, the patient developed pain in the left side of the neck, and began having disturbances of equilibrium, with headache and vomiting. A mastoid operation was performed and the antrum found filled with granulation and pus. The sinus was uncovered but appeared to be normal. In a few days, signs of a posterior cerebellar syndrome were observed and the patient became delirious and entered a state of coma. Vestibular examination showed no nystagmus, but definite reaction to caloric test, marking the abscess as definitely posterior in variety. The cerebellar dura was, therefore, exposed, as was the lateral sinus still further, and tapping behind the sinus resulted in withdrawal of a considerable amount of pus. In addition, an intrasinal abscess was discovered, presumably the starting point of the cerebellar abscess. A large incision was made into the sinus and united with the horizontal incision for tapping the cerebellar abscess, a drain being inserted at this point. Following one period of temporary interference with drainage, the patient made a rapid and uneventful recovery. The author stresses the fact that diagnosis was relatively easy because of the signs of cerebellar involvement, with coma and stiffness of the neck. Localization was facilitated by the absence of nystagmus and the presence of caloric reaction, there being no signs of vestibular deficiency. The abscess probably originated from a thrombosis in the lateral sinus in spite of its negative appearance at the first operation. Only by laying it more widely open and tapping it was pus discovered in a latent abscess of the thrombosed sinus. Had an ordinary routine been pursued, the abscess might possibly have been tapped anterior to

the sinus and uninvolved normal cerebellar tissue encountered. Risk of opening the pons cerebelli would have been present. Failure to explore the lateral sinus might have resulted in failure to discover the latent thrombosis and a mistaken understanding of the origin of the abscess might have resulted in an operation on the labyrinth, with obvious futility. The author concludes that labyrinthine examination facilitates localization of the site of the abscess and that anterior cerebellar abscess of labyrinthine origin is due to complications arising in the labyrinth itself. The prognosis is serious and drainage must be made anterior to the sinus following labyrinthine operation. A posterior cerebellar abscess has no labyrinthine symptomatology and is usually due to thrombosis of the lateral sinus. Its prognosis is less serious and it must be treated by an incision behind the sinus without the necessity of any labyrinthine intervention. Wide sinus exposure is important.

CONCLUSIONS.

If one may venture to summarize a summary, it is apparent to this reviewer, from the above outline of the major literary contributions to the subject of otitic sepsis for 1939, that startling and gratifying progress is being made in dealing with these heretofore serious and ofttimes discouraging clinical problems. The advent of sulfanilamide and sulfapyridine is obviously already leading to recovery in many cases which were formerly regarded as hopeless situations. Uncertainty and disagreement still are evident with respect to the timing of these drugs, the indications for their employment, particularly at the outset of otitic infection, and with respect to details as to dosage, methods of administration and rules for continuation or withdrawal. Improved operative techniques particularly for approaching the petrous pyramid and in dealing with brain abscess are obvious in almost every article dealing with these conditions. Stimulated by these successes, there is no question that more intensive study and still further progress will be made along otological fields which were thought some years ago to have been explored to their fullest extent. Otologists may well be gratified with their achievements and may look forward to still greater heights of attainment.

BIBLIOGRAPHY.

1. Grove, W. E.: Nursing, Feeding and Parenteral Administration of Fluids. *Arch. Otolaryngol.*, 30:872-875, Dec., 1939.

2. Sutherland, J. M.: Transfusions of Blood in the Care of Patients After Operations for Otitic Sepsis. *Arch. Otolaryngol.*, 30:876-900, Dec., 1939.

3. Galloway, T. C.: Chemotherapy. *Arch. Otolaryngol.*, 30:901-915, Dec., 1939.

4. Juers, A. L.: Intracranial Complications in 250 Cases of Surgical Mastoid Infections. *Ky. Med. Jour.*, 37:585-588, Dec., 1939.

5. Aries, P. L.: Pediatric Treatment of Otologic Sepsis, Including Meningitis. *Ill. Med. Jour.*, 75:131-136, Feb., 1939.

6. Maybaum, J. L., and Druss, J. G.: Otitic Infections Due to Pneumococcus Type III. *Arch. Otolaryngol.*, 30:21-37, July, 1939.

7. Long, P. H.; Bliss, E. A., and Feinstone, W. H.: Mode of Action, Clinical Use and Toxic Manifestations of Sulfanilamide. *Jour. A. M. A.*, 112:115, Jan. 14, 1939.

8. Long, P. H.: Sulfapyridine. *Jour. A. M. A.*, 112:538, Feb. 11, 1939; 112:1850, May 6, 1939.

9. Long, P. H.; Bliss, E. A., and Feinstone, W. H.: Effects of Sulfapyridine, Sulfanilamide and Related Compounds in Bacterial Infections. *Pa. Med. Jour.*, 42:483-491, Feb., 1939.

10. Powell, H. M., and Chen, K. K.: Action of Sulfapyridine. *Jour. Pharmacol. and Exper. Therap.*, 67:79-100, Sept., 1939.

11. Fenton, R. A.: Sulfanilamide in Otolaryngology. *Ann. Otol., Rhinol. and Laryngol.*, 48:17, March, 1939.

12. McLaurin, J. G.: Sulfanilamide in Otolaryngology. *Ann. Otol., Rhinol. and Laryngol.*, 48:23, March, 1939.

13. Cunning, D. S.: Treatment of Otitic Meningitis. *Arch. Otolaryngol.*, 30:950-972, Dec., 1939.

14. Eggston, A. A.: Bacteriology of Suppurative Meningitis. The Laryngoscope, 49:16-20, Jan., 1939.

15. Dwyer, J. G.: Differential Diagnosis. The Laryngoscope, 49:21-22, Jan., 1939.

16. Jones, M. F.: Avenues of Infection. The Laryngoscope, 49:23-25, Jan., 1939.

17. Bowers, W. C.: Operative Treatment of Meningitis. The Laryngoscope, 49:26-29, Jan., 1939.

18. Appelbaum, E.: Chemotherapy in Meningitis, Secondary to Ear and Sinus Infections. The Laryngoscope, 49:30-45, Jan., 1939.

19. Falor, W. H.: Betahemolytic Streptococcic Meningitis Treated with Sulfanilamide; Case. *Ohio State Med. Jour.*, 35:154-156, Feb., 1939.

20. Goodyear, H. M.: The Indication for Surgery in Meningitis Secondary to Disease of the Middle Ear and of the Nasal Sinuses; Reservations in Early Surgical Intervention. The Laryngoscope, 49:102-105, Feb., 1939.

21. McCaskey, C. H.: The Treatment of Otitic Meningitis Due to Streptococcic Infection by Surgery and Sulfanilamide. *Surg., Gynecol. and Obstet.*, 68:377-389, Feb., 1939, No. 2A.

22. MAYBAUM, J. L.; SNYDER, E. R., and COLEMAN, L. L.: Experiences with Sulfanilamide Therapy for Otogenous Infections, with Special Reference to Masking of the Clinical Course. *Arch. Otolaryngol.*, 30:557-575, Oct., 1939.

23. DOANE, J. C.; BLUMBERG, N., and TEPLICK, J. G.: Sulfanilamide Therapy. A Review of Literature. With a Report of Two Cases of Streptococcic Hemolytic Meningitis with Recovery. *Med. Rec.*, 150:439-443, Dec. 20, 1939.

24. HAWTHORNE, A. T.: A Case of Streptococcus Meningitis of Otogenic Origin with Recovery. *Va. Med. Mon.*, 66:267-269, May, 1939.

25. NEAL, J. B.: Meningitis. *Pa. Med. Jour.*, 42:1019-1027, June, 1939.

26. YULE, A. P.: Pneumococcus Meningitis, Following Mastoidectomy, Treated with M and B 693 (Sulfapyridine, Sulfanilamide Derivative); Recovery. *Brit. Med. Jour.*, 1:872-873, April 29, 1939.

27. CAVENAGH, J. B.: Pneumococcal Meningitis of Middle Ear Origin. A Case of Recovery. *Jour. Laryngol. and Otol.*, 54:624-626, Oct., 1939.

28. HALL, I. S.: Acute Labyrinthitis and Meningitis: Recovery. Societies' Proceedings. The Scottish Otological and Laryngological Society. *Jour. Laryngol. and Otol.*, 54:211-213, April, 1939.

29. THOMAS, A.: Purulent Meningitis Produced by Minute Hemolytic Streptococcus (Long and Bliss) and Bacillus Coli. *Jour. Mt. Sinai Hosp.*, 5:702-704, March-April, 1939.

30. ROSENWASSER, H.: Otitic Streptococcus Meningitis with Recovery. *Jour. Mt. Sinai Hosp.*, 6:203-205, Nov.-Dec., 1939.

31. PHILIPSON, J.: Two Cases of Otitic Streptococcal Meningitis with Recovery. *Acta Oto-Laryngol.*, 27:654-661, 1939.

32. MICHELS, M. W., and GONNE, W. S.: Meningitis Due to Staphylococcus Aureus Hemolyticus Treated with Sulfanilamide. *Amer. Jour. Dis. Child.*, 57:1379-1385, June, 1939.

33. CANUYT, G.: Acute Meningitis Treated by Extensive Surgical Intervention and Sulfanilamide: Four Cases with Recovery. *Ann. d'otolaryngol.*, pp. 654-661, July, 1939.

34. CANUYT, G.: Acute Puriform Meningitis of Otogenous Origin Late After Cranial Trauma with Trephining; Surgical Intervention and Sulfapyridine (Sulfanilamide Derivative) Therapy; Case with Recovery. *Ann d'otolaryngol.*, pp. 662-664, July, 1939.

35. ESCAT, M.: Meningitis Treated by Sulfanilamide; Two Cases. *Rev. de laryngol.*, 60:411-414, May, 1939.

36. OMBREDANNE, M., and PAPAIOANNON, A.: Streptococcic Meningitis, Case with Recovery After Sulfanilamide and Surgical Therapy. *Bull. Soc. Pediat. de Paris*, 37:116-122, March, 1939.

37. BUCKMAN, S. T.: Hemolytic Streptococcic Meningitis of Otitic Origin in a Young Woman at Term: Recovery and Delivery of a Living Child. *Arch. Otolaryngol.*, 29:853-856, May, 1939.

38. BROWN, J. E.; EMSWILER, H. A., and RECK, L. E.: Influenzal Meningitis with Recovery: Treatment with Mastoidectomy, Sulfanilamide and Continuous Spinal Drainage. *Arch. Otolaryngol.*, 29:860-864, May, 1939.

39. Teed, R. W.: Streptococcic Meningitis with Recovery. *Arch. Otolaryngol.*, 29-716-719, April, 1939.

40. WHITE, L. L.: Otitic Meningitis Successfully Treated with Sulfanilamide. *Arch. Otolaryngol.*, 29:159-160, Jan., 1939.

41. Gonne, W. S.; Michels, M. W., and Watson, T.: Recovery from Hemolytic Streptococcic Meningitis of Otitic Origin. *Arch. Otolaryngol.*, 29:382-387, Feb., 1939.

42. Silverman, D., and Thorner, M.: Treatment of Meningitis Due to Pneumococcus Type III, with Sulfanilamide; Recovery. *Arch. Otolaryngol.*, 30:431-436, Sept., 1939.

43. Shea, John J.: What Is Meant by Petrositis? *Memphis Med. Jour.*, 14:9:160, Sept., 1939.

44. Towson, C. E.: Petrositis: Report of a Case with a Review of Some of the Literature. *Arch. Otolaryngol.*, 29:468-479, March, 1939.

45. Lillie, H. I., and Williams, H. L.: Chronic Suppurative Lesions of the Petrous Pyramid: Report of Six Cases with Different Pathologic Characteristics. *Arch. Otolaryngol.*, 29:345-355, Feb., 1939.

46. Kopetzky, S. J.: Progress in Otolaryngology. Purulent Otitis Media, Mastoiditis, Sinus Thrombosis and Suppuration of the Petrous Pyramid. *Arch. Otolaryngol.*, 30:800-829, Nov., 1939.

47. Gerlings, P. G.: Petrositis with Suppurative Meningitis. Ramadier Operation. Recovery. *Jour. Laryngol. and Otol.*, 54:471-475, Aug., 1939.

48. Asherson, N.: A Case of Petrositis, Stimulating Cellulitis. *Jour. Laryngol. and Otol.*, 54:485-486, Aug., 1939.

49. Voss, O.: Kanmman Pyramiden-Eiterungen Vorbeugen? *Acta Oto-Laryngol.*, 27:498-504, 1939.

50. Jones, M. F.: Care of Mastoid Wounds Following Complete Mastoid Operation, Sinus Thrombosis and Operations on the Petrous Pyramid. *Ann. Otol., Rhinol. and Laryngol.*, 48:579-584, Sept., 1939.

51. Bateman, G. H.: Petrositis: Operation and Recovery. A Report of Two Cases. *Jour. Laryngol. and Otol.*, 54:476-484, Aug., 1939.

52. Frenckner, P.: Indications for Labyrinthectomy in Cases of Labyrinthitis. *Acta Oto-Laryngol.*, 27:19-36, 1939.

53. Layton, T. B.: Treatment of Meningitis. Societies' Proceedings. Royal Society of Medicine, Section on Otology. *Jour. Laryngol. and Otol.*, 54:364, June, 1939.

54. Cody, C. C., Jr.: Thrombosis of the Lateral Sinus. *Arch. Otolaryngol.*, 29:674-680, April, 1939.

55. Joannovich, D.: Therapeutic Results in 30 Cases of Otogenous Sepsis. *Ztschr. f. Hals., Nasen. u. Ohrenheilk.*, 45:17-21, 1939.

56. Jones, M. F.: Care of Mastoid Wounds Following Complete Mastoid Operation, Sinus Thrombosis and Operations on the Petrous Pyramid. *Ann. Otol., Rhinol. and Laryngol.*, 48:579-584, Sept., 1939.

57. Ersner, M. S.: Otitis Media and Mastoiditis Simulating Thrombophlebitis of the Lateral Sinus. *Arch. Otolaryngol.*, 29:605-613, April, 1939.

58. Goldman, J. L.: Blood Cultures in Cases of Otitic Sepsis. *Arch. Otolaryngol.*, 30:516-524, Oct., 1939.

59. Druss, J. G.: Thrombosis of the Lateral Sinus: A Study of 15 Cases, Including Histologic Examination. *Arch. Otolaryngol.*, 29:42-64, Jan., 1939.

60. Smith, M. T.: Thrombophlebitis of the Lateral Sinuses Complicating Bilateral Acute Mastoiditis. *Arch. Otolaryngol.*, 29:533-543, March, 1939.

61. Gerzog, B. G.: Bacillus Proteus in Otogenic Infections, Secondary Mastoiditis, Thrombosis of the Lateral Sinus and Bacteremia. *Arch. Otolaryngol.*, 30:275-279, Aug., 1939.

62. Schwartzman, G.: Immunologic Aspects of Blood Invasions with Special References to Thrombosis. The Laryngoscope, 49:653-665, Aug., 1939.

63. Adelstein, L. J.: Surgical Management of Otogenous Abscess of the Brain. *Arch. Otolaryngol.*, 29:624-639, April, 1939.

64. Alpers, B. J.: Abscess of the Brain: Relation of the Histologic to the Clinical Features. *Arch Otolaryngol.*, 29:199-222, Feb., 1939.

65. Kahn, E. A.: Treatment of Encapsulated Abscess: Visualization by Colloidal Thorium Dioxide. *Arch. Neurol. and Psychiat.*, 41:158-156, Jan., 1939.

66. Krainz, W.: Otogenic Abscess: 11 Cases. *Monatsschr. f. Ohrenheilk.*, 73:599-605, Sept., 1939.

67. Kulenkampff, D.: Otogenic Abscess and Its Treatment. *Deut. med. Wchnschr.*, 65:1311-1313, Aug. 18, 1939.

68. Sjoberg, A. A.: Contribution to Prognosis and Treatment of Rhinogenous and Otogenous Brain Abscesses. *Acta Oto-Laryngol.*, 27:638-653, 1939.

69. Laskiewicz, A.: Les Abces Cerebrauz d'origine otique et nasale. *Acta Oto-Laryngol.*, Supp. 37, 1-114, 1939.

70. Tobey, H. G.: Care of Mastoid Wounds Following Operations on Abscess. *Ann. Otol., Rhinol. and Laryngol.*, 48:590-595, Sept., 1939.

71. Tempea, V.: Meningitis with Cerebral Abscess: Case with Recovery After Surgical Therapy. *Ann. d'otolaryngol.*, pp. 243-246, March, 1939.

72. Van den Wildenberg: Cerebellar Abscess of Otitic Origin in Child Six Years Old; Case with Recovery After Surgical Therapy. *Ann. d'otolaryngol.*, pp. 550-553, June, 1939.

73. Aubry, M.: Posterior Abscess: Case. *Ann. d'otolaryngol.*, pp. 485-489, May, 1939.

PRESIDENT'S ADDRESS.

OBSERVATIONS ON THE CONSERVATION OF HEARING.*

DR. HORACE NEWHART, Minneapolis.

Achievement in the prevention and alleviation of conditions causing hearing deficiencies in comparison with attainment in preventive work in other fields, as ophthalmology, until recently has been embarrassingly slow. Progress has been so retarded as to cause disappointment and reproachful criticism of the otologist by the aurally handicapped and those interested in their problems.

Under these conditions it is appropriate that we give frank consideration to some of the aspects of the problem of conserving the hearing to which the busy otologist as a rule has given little serious thought.

The past decade has witnessed creditable accomplishment in this field of preventive medicine. The success thus far attained has been relatively sporadic and has served largely to disclose the possibility for greater achievement. It emphasizes the need for greatly increased activity under the leadership of especially qualified physicians if we would achieve a degree of success commensurate with the educational, economical and social importance of the problem whose magnitude is indicated by the surprisingly high incidence of hearing defects among our population.

In the past there have been extenuating circumstances which have retarded progress in this field. To mitigate our embarrassment and for a better understanding of the problem we mention the following:

1. The anatomical inaccessibility of the ear for research, diagnosis and treatment.

*Read before the Seventy-third Annual Meeting of the American Otological Society, Inc., Rye, N. Y., May 30, 1940.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Aug. 22, 1940.

2. The inadequacy of the classic hearing tests for the accurate detection and measurement of hearing deficiencies.

3. The persistence of the widespread belief that efforts to prevent, cure or compensate satisfactorily for handicapping hearing defects are for the most part futile. This idea, which formerly was justified by the limitations of our knowledge, has resulted in lamentable neglect of the ears and is largely responsible for a general indifference toward organized effort to conserve the hearing.

4. There has existed among physicians, educators and the public a conspicuous lack of familiarity with more recently acquired fundamental facts pertaining to the subject. These facts are not sufficiently stressed in the undergraduate teaching of otology in many medical schools and are given scant attention in public school health programs and in popular and professional courses on preventive medicine and public health. One of the most important of these omitted facts is that many individuals have unrecognized handicapping hearing impairments. This is especially true among younger school children in whom speech defects, retardation, inferiority complexes and emotional and social maladjustments are caused in many cases by slight but very significant hearing deficiencies. This should be impressed on every physician, teacher and parent.

We would broadly define a significant hearing deficiency as any departure from normal hearing acuity which is capable of interfering with the natural acquisition of articulate speech, a fair education and a personality which will insure to the individual his economic and social security and the enjoyment of a reasonably successful life. This includes potentially handicapping as well as already handicapping deficiencies.

The most notable impetus to progress in the solution of the problem of conserving the hearing has been through the employment of the audiometer in research, clinical practice and the early detection of hearing loss.

Research in recent years has disclosed many previously unrecognized causes of hearing impairment. This has correspondingly increased the possibility of their successful prevention and treatment. The exact proportion of cases of

hearing deficiencies which can be prevented, cured, improved or arrested cannot be readily determined by laboratory methods because of the great amount of time and clinical material required. Nevertheless, on the basis of extensive clinical experience gained by competent observers, we now know the number to be large and rapidly increasing. In the opinion of many otologists it amounts to a majority of all who ultimately seek otological aid, in many instances too late to obtain satisfactory results from treatment.

We would briefly consider certain fundamental facts which must be better understood and consistently applied by members of the medical profession if we would rapidly advance the cause of better hearing.

It is recognized that among various measures now available, the one most effective means for reducing on a comprehensive scale the high incidence of hearing defects among our population is by incorporating in every public school health program the periodic testing of the hearing of all pupils by scientifically approved audiometers. This procedure makes possible the early detection of hearing defects and the prompt application of preventive and corrective measures.

Today we can safely predict that ultimately, when their advantages are fully appreciated, these tests will be routinely made of the registrants in all educational institutions and of the members of industrial, military, naval and various civic groups. Already they are finding wide application in aviation and in the health programs of a number of the progressive industrial organizations in America.

As related especially to public schools this expanded health program provides that each pupil found to have a significant hearing deficiency will receive through the proper agencies an otological examination to make possible prompt corrective medical care, educational adjustment, rehabilitation and vocational training as needed.

In several States this procedure has been made mandatory by law. Where this has not been done we would emphatically stress the point that the initiative necessary to secure its introduction and its proper administration should come from otologists and interested, informed medical practitioners through the exercise of their recognized function as leaders

in popular medical education. This must always be done most tactfully and in close co-operation with the local school, public health and welfare organizations.

Objection is occasionally made by uninformed physicians that the use of audiometer tests in the public schools is a step toward socialized medicine and an invasion by the educational authorities of private practice. On the contrary, the discovery of unsuspected hearing defects definitely increases the work of the private practitioner who is interested and capable of rendering service in this field. No attempt on the part of the school nursing personnel to diagnose or treat ear conditions is to be tolerated under the medical practice acts of all of our States.

Although several States and hundreds of local units in the United States and in some foreign countries already have put this procedure into successful operation, to date the field has been only slightly covered, particularly in rural areas where the need has been shown to be especially great. It is encouraging to note that during the school year 1938-39, 1,900,000 pupils in the United States were given audiometer tests. This is an encouraging increase over previous years.

The instrument most generally used for screening the school population to discover those having auditory deficiencies has been the Western Electric phonograph audiometer. With it groups of as many as 40 pupils are tested simultaneously. Pupils discovered to have an apparent deficiency are retested, preferably by a pure tone or pitch range audiometer. Those who on retest still show a significant hearing loss are classed as needing referral to an otologist or physician for an otological examination.

Tests with the phonograph audiometer during the past 15 years have proved vastly more accurate, time-saving and economical than tests by any previously used methods. The procedure has been generously indorsed by federal agencies by grants of large sums to finance hearing surveys covering entire States and selected communities.

Serious criticism has been made of these surveys because of the failure on the part of local agencies to provide an adequate medical follow-up of hearing-deficient children to insure needed care for the underprivileged and medically indigent.

This is a problem which can be solved only by co-operation among local school, medical and welfare organizations in a plan which will provide the best possible otolaryngological service with equal fairness to both the recipients and the medical profession.

In order that screening tests be most effective and reliable, the limitations of the phonograph audiometer, in spite of its marked superiority over the older means of making hearing tests, should be known to physicians, educators, public school nurses and others:

1. This type of instrument cannot be used advantageously for the rapid, simultaneous testing of large groups of children who are too young to write dictated numbers. These pupils constitute an important part of the school population who frequently are not tested because of the increased time required. It is possible, however, to test them less rapidly with the additional help of monitors to whom the numbers heard are whispered. Individual tests by "accepted" pure tone audiometers yield more accurate results.

2. The phonograph audiometer fails to disclose hearing impairment for the higher frequencies. Ability to hear tones of higher pitch recently has been found to be important for the early diagnosis of impaired tubal function and for the acquisition of correct speech. Individual tests by the pure tone audiometer reveal many more cases of significant loss than are disclosed by the phonograph audiometer.

The inconsistently wide variations in the incidence of hearing defects as occasionally reported by different observers in different communities has tended to discredit the use of the phonograph audiometer and, in some instances, has delayed its adoption. These discrepancies are explained by frequent failure to eliminate distracting and masking noises and to follow closely the operating instructions of the manufacturers. The lack of uniform standards for evaluating the findings is also a serious obstacle to obtaining scientifically accurate, comparable results.

The early, authoritative establishment of such standards is an urgently needed task to be undertaken by the otologist and physicist. Incidentally, it should be stated in its defense that the phonograph audiometer was not designed as an

instrument of precision; nevertheless, it has rendered a most valuable service.

We would emphasize the fact that school hearing tests as made by nurses or technicians should be under the direction of qualified physicians who are familiar with the technique and clinical significance of audiometric testing. Instruction in the best methods of making modern hearing tests for screening purposes are offered in a number of teachers colleges and training schools for nurses.

Phonograph audiometers for group testing utilizing pure tones instead of the voice are being developed, but to date have not been extensively used. Certain limitations must be overcome before they are likely to supersede instruments now employed.

The recent work of Dr. Crowe and Dr. Guild and their associates, whom we are most happy to have on our program today, suggests the necessity of early revision of the techniques now used in making hearing tests, especially of younger individuals. Such revision must be based on extensive research now well under way. Until this revision shall have been perfected we would urge the continued and wider use of the audiometers now employed.

In response to a growing demand for simple, pure tone audiometers designed especially for expeditiously screening larger groups of school children and others by individual tests, instruments having a fixed zero threshold level which permits rapid operation have been made available at low cost.

To determine their practicability, tests were recently made under our direction by the two experienced nurse-audiometrists of the Minneapolis Public Schools. A total of 6,344 pupils from the second to the sixth grades, inclusive, were tested between September, 1939, and April, 1940. Two Maico D6 pure tone school audiometers having discrete frequencies from 128 cycles per second to 8,192 cycles at octave intervals were employed. A deficiency of 15 or more decibels at one or more frequencies was arbitrarily considered a significant defect. All tests were made in relatively quiet schoolrooms.

Of the 6,344 tested, 5,084, or 80.2 per cent, were rated as normal.

In 1,260, or 19.8 per cent, deficiencies were found. Of these, 916, or 14.4 per cent, had losses only for the two higher frequencies of 4,096 and 8,192 cycles; and 344, or 5.4 per cent, had losses only for the frequencies below 4,096 cycles. The high frequency losses occurred 2.67 times as often as those of lower pitch.

These preliminary findings, in our opinion, clearly indicate the frequent occurrence of impaired perception for high pitched tones among younger individuals and prove the greater accuracy and desirability of individual tests by audiometers of this type.

We recommend that at least one tone of a frequency above 8,192 cycles, as 11,548 cycles, be included in these school tests for screening purposes. It should be noted that in testing with 8,192 cycles and higher frequencies, there is present the possibility of error due to the shortness of the wave lengths, since slight variations in the distance between the tympanic membrane and the receiver diaphragm cause marked differences in the loudness of the test tones. Thus faulty placing of the receiver over the auricular canal and changes in the pressure with which the receiver is held against the external ear may cause the tone to become audible or inaudible. To avoid this, special care is necessary.

From what has been stated, perhaps in too great detail for this advanced group, it should be evident that there is serious need and a large opportunity for further research for the determination of the incidence, causes and clinical significance of hearing deficiencies among the school population. This must be based upon the establishment of uniform standards for the practical application of screening and diagnostic audiometer tests which it is hoped will soon be made.

There exists also a keenly felt need for the adoption of acceptable, standardized audiogram blanks and uniform symbols for recording audiometric findings so that an audiogram made by one observer may be easily interpreted by others. This task might appropriately be undertaken by the Committee on Audiometers and Hearing Aids of the American Medical Association, subject to the approval of our national otolaryngological societies.

We would recommend for the serious consideration of our members and others interested in our specialty the following

as important steps for greater accomplishment in otologic practice.

1. The early inclusion in the teaching of otology to undergraduates of fundamental information concerning:

a. The technique for the discovery and measurement of hearing defects;

b. The prevention, correction and amelioration of hearing handicaps by modern methods;

c. The importance of preserving residual hearing and supplementing it by carefully prescribed hearing aids and lipreading; and

d. Provisions for social and economic readjustment of the aurally handicapped through local and State agencies.

2. The early revision of the terminology of otology in accordance with modern conceptions of the subject. As a conspicuous instance of this need we mention the very general abuse of the words *deaf* and *deafness.* Thoughtlessly and by example of long usage we still carelessly employ these terms indiscriminately in referring to all degrees of hearing impairment from a total loss of function of both ears to a slight recognizable deficiency affecting possibly only one ear. This is highly confusing and in many instances implies a certain unfair stigma and causes a feeling of inferiority which is justly resented by persons having relatively slight defects. The use of these terms should be rigidly restricted to indicate only the most severe degrees of hearing loss. For designating all lesser degrees we will more appropriately use instead such terms as *hard-of-hearing, hearing deficient, hearing deficiency, defect* or *impairment.* We do not call persons with visual deficiencies correctible by glasses *blind,* nor do we refer to their defects as *blindness.*

The Committee on Nomenclature of the Conference of Executives of Schools for the Deaf has anticipated the otologists by adopting for their needs definitions for *deaf* and the *hard-of-hearing* as follows:

"*1. The Deaf:* Those in whom the sense of hearing is nonfunctional for the ordinary purposes of life; divided into two distinct classes based on the time of the loss of hearing.

"*a.* The congenitally deaf.

"*b.* The adventitiously deaf — those who were born with normal hearing but in whom the sense of hearing became nonfunctional later through illness or accident.

"*2. Hard-of-Hearing:* Those in whom the sense of hearing, although defective, is functional with or without a hearing aid."

We would recommend that this Society, having today arrived at a sufficiently mature age, appoint a committee on nomenclature to initiate steps toward bringing our terminology up to date, as is being done by other national medical groups.

Among the many fields still wide open for research which should appeal to members of this Society we would mention as of special importance the increasing number of hearing hazards in industry. Investigation of the incidence, causes and means of preventing hearing impairment due to known and unrecognized factors in the environments of workers in many newly developed industrial fields will result in a material reduction in the number of aurally handicapped adults engaged in industrial pursuits.

527 Medical Arts Building.

THE GENERAL PROBLEM OF DEAFNESS IN THE POPULATION.*†

DR. WILLIS C. BEASLEY, Bethesda, Md.

INTRODUCTION.

If impaired hearing were an infectious disease, the present topic could be characterized with historical accuracy under the title, "Some Epidemiological Aspects of Deafness." Many of the principles underlying epidemiological observation and analysis which have been applied to the study of diseases, whether of infectious or noninfectious origin, as mass phenomena are utilized in this study of impaired hearing in the general population. In epidemiological observation the time of onset, periodicity or temporal course, demographic distribution, rate of prevalence and rate of incidence of infectious diseases are studied among large and diversified population groups rather than among clinically isolated individuals. The purpose in broad terms of epidemiological investigation is to discover biological features of disease that cannot be revealed through clinical observation alone. Although the objectives of epidemiological study are unique, it is dependent intimately upon the specific knowledge derived from clinical medicine, especially from bacteriology, and upon the methods for treating data arising under the province of vital statistics or, better, biostatistics. Knowledge of the clinical manifestations and reactions of a disease is fundamental to effective epidemiological research; but it is only through observing the reactions of specific micro-organisms upon diversified and extensive populations under complex living conditions that deductions can be made regarding such detailed characteristics as: *a.* the *infectivity* and *pathogenicity* of bacteria; *b.* the *resistance* of the human organism to infection and pathogenic effects; and *c.* the biological and environmental factors responsible for variations in resistance of human organisms, either naturally possessed or arti-

*Read by invitation at the Seventy-third Annual Meeting of the American Otological Society, Inc., Rye, N. Y., May 30-31.

†From the Division of Public Health Methods, National Institute of Health, U. S. Public Health Service, Bethesda, Md.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Sept. 1, 1940.

ficially acquired. Inasmuch as all epidemiological investigation deals with quantitative data that are affected by a multiplicity of factors, proper evaluation of such observations must include rigorous statistical analysis.*

Centuries of experience in the epidemiological study of diseases have provided fundamental facts and principles which constitute epidemiology as a basic division of public health science; but, equally important, the results of this experience have led to a conception of epidemiological technique as a method of attacking a wide variety of public health problems, even though the immediate object of investigation is neither the course of a particular infectious disease nor the demographic reactions of specifically isolated micro-organisms. It is true, of course, that in the early history of epidemiological study *infectious* diseases constituted the objects of investigation — hence, etymologically, the concept of "epimedic diseases" and, consequently, the science of "epidemiology." In recent years, lasting contributions to medical science have been achieved through truly epidemiological study of tuberculosis and cancer. Still more recently, valuable discoveries have been made through epidemiological study of dental caries. Thus, epidemiology apparently is destined to embrace investigations dealing not only with infectious diseases but also with any kind of tissue morbidity, either general or localized. Through a simple logical extension of this thought, *functional* morbidities of striped or smooth muscles, bone tissue, nerve processes or of sense organs may be included among the processes of organisms concerning which further knowledge can be attained by application of epidemiological methods of investigation.

Application of epidemiological technique to a study of deafness in the general population is a logical outgrowth of this broadened point of view. Impaired hearing is a functional morbidity, varying in degree and type, which arises from a wide variety of causal or predisposing factors, such as morphological and physiological constitution, physical environmental conditions, nutritional deficiencies, systemic and localized infections, endocrine abnormalities and toxic drugs. The clinical manifestations of hearing deficiencies are spe-

*For further elucidation of these general principles, the reader is referred to Greenwood (36, 37), and especially to Frost's (34) excellent discourse on the philosophy of epidemiological method.

cific for only a few types of deafness; for others, the exact etiology is still unknown or under debate; and indications for treatment, either preventive or arrestive, are lacking for several different types of cases. Even physical therapy, the prescription of artificial hearing aids, has not been carried to a satisfactory stage of development. It is clear that only through rigorous prosecution of clinical study can progress be made on many phases of etiology, diagnosis and prognosis. Symptomatology is lacking in coherence and definitive classification. At the present, a consideration of theoretically possible and some actual contributions from epidemiological study of impaired hearing is timely, in view of the failure of clinical observation to supply much of the needed information.

Knowledge concerning the rate of prevalence and the rate of incidence of various types and degrees of impaired hearing in broad demographic groups constitutes the basic information for deducing the existence of significant trends, which may in turn lead to the discovery of causal or predisposing factors. Variations between rates of prevalence among different age, sex, racial, economic, occupational and geographic groups (and these studied in relation to secular, seasonal, nutritional, climatic and other factors) are signals indicating differential *causal* factors are operative, and point the way for detailed and intensive clinical investigation. When observations on such differential trends in rates of prevalence are supplemented by clinical studies of properly chosen samples of larger groups, identification of etiological factors, which otherwise could not be isolated, is greatly facilitated; however, in the absence of a fairly comprehensive epidemiological study, even basic data on annual incidence of new cases at various ages remains largely conjecture, as in the past. Aside from the study to be reported at this time, there are no investigations of a similar nature from which at least a first approach to an epidemiology of impaired hearing can be made.

An extensive review (Beasley[1]) of available information on prevalence of any clearly defined or identified degree of impaired hearing in the population indicates definitely that our knowledge is limited to data collected on very special and limited population groups. These are as follows: *a.* surveys in the public schools; *b.* enumeration of deafmutes by the U. S.

Bureau of the Census; *c.* cumulative records on hospital patients; *d.* observations on army recruits during the operation of the Selective Service Act prior to Dec. 15, 1917. The character of these data is such that they are unrelated to each other in manner of collection and definitions or criteria employed. It is impossible to derive estimates on rates of prevalence in the general population from these sources, except for the age range of children in the public schools (under 15 years).

This report brings together a related set of data which was collected by the United States Public Health Service during the National Health Survey (1935 and 1936) and through a special clinical investigation of hearing (1936), which supplemented the household census. In view of the complexity and abundance of materials from these two sources, especially the clinical study, only certain phases of the data bearing on broad epidemiological aspects are included in this paper. Specifically, consideration is given to: *a.* prevalence and annual incidence of *severely impaired* hearing in relation to age, sex, family income, employment status and occupational classes; and *b.* prevalence and annual incidence of progressive high-tone deafness involving minor and moderate impairment for perception of conversational speech in relation to age and sex. Special attention is given in this section to the problem of symptomatic loss of hearing for high tones in childhood, and the predominating trend of chronic progressive nerve deafness in adult years. For, at the present, a relation between the two phenomena has not been demonstrated, even though usually it is assumed that the two are dependent. It is planned to present in a subsequent report an analysis of cases according to differential diagnosis of conductive and nerve lesions, in relation to age, sex, time of onset, duration of impairment since first noticed, occupation and findings from a rather thorough otological examination.

THE NATIONAL HEALTH SURVEY.

Students of public health have realized for a long time that many problems in formulating an adequate national policy could never be solved until at least a descriptive* epidemiological study of current acute illnesses and chronic diseases

in a large sample of the general population was carried out. This objective dominated the planning and conduct of the National Health Survey. In this investigation, information was collected on current disabling illnesses, chronic diseases and physical impairments in relation to a broad range of socioeconomic factors (Perrott, *et al.*, 55; Britten, *et al.*, 12). In addition to the basic information on sickness, extensive facts were obtained which serve to identify personal characteristics of individuals, as well as living conditions of families in a material sense. These include age, sex, race, occupation, employment status, marital status, total family income, size of family, composition of household and famly, housing conditions and other similar data.

In securing information about physical impairments, enumerators, who had been trained thoroughly in the task of interviewing, inquired as to the condition of hearing for each member of a household and, on the basis of replies to a standard series of questions, classified the type of impairment in one of the folowing five groups:

1. Partial deafness, stage 1: The individual has difficulty in understanding speech in church, at the theatre or in group conversation, but can hear speech at close range without any artificial assistance.

2. Partial deafness, stage 2: The individual has difficulty hearing direct conversation at close range, but can hear satisfactorily over the telephone or can hear loudly spoken speech.

3. Partial deafness, stage 3: The individual has difficulty hearing over the telephone at ordinary intensities, but can hear amplified speech by means of hearing aids, trumpets or other means of amplification.

4. Total deafness for speech: The individual cannot hear speech under any circumstances, but acquired the hearing defect after learning to speak language by ordinary means.

*A distinction is presumed here between descriptive and direct epidemiological observation. The direct method is based on clinical observation, instead of social and medical history alone. Descriptive data of the latter type only were collected during the National Health Survey. A nationwide health survey, based on direct clinical observation, would constitute an epochal contribution to medical and public health science.

5. Deafmute: The individual was born deaf or acquired severe deafness sufficiently early in life to prevent him from learning speech through the usual means.

All persons who were not designated on the schedule as having a hearing impairment, in accordance with one of these five classifications, became characterized in the final analysis as having normal hearing.* This so-called "normal hearing" group, of course, is composed of persons *a.* actually regarded by the informant as not qualifying under any of the stages of deafness as defined; *b.* persons having impairments of hearing not known to the informant; and *c.* persons having impairments that were known to the informant but which were not reported to the enumerator for various reasons. It is clear that in this "normal hearing" group there are many cases of impaired hearing, ranging from minor to severe levels of hearing loss, for which no count was obtained in the household census tabulations; therefore, the actual prevalence of impaired hearing in both the Health Survey population sample and the whole population must be significantly higher than the count obtained from the household census. In the supplementary clinical study, an attempt was made to evaluate this source of error in the household census.

CHARACTERISTICS OF THE SAMPLE.

In evaluating the extent to which the present data may be regarded as representative of the general population, it is necessary to consider the procedure employed in determining the original Health Survey population sample. In the first place, the population upon which this report is based is entirely urban. There are good theoretical reasons for supposing, but no convincing evidence, that the prevalence and character of impaired hearing may differ significantly for rural and urban populations. The National Health Survey covered only a relatively small and nonrepresentative rural population. Consequently, data on impaired hearing in rural areas are not considered in this report.

For purposes of sampling, the country was divided into four geographic areas, as follows:

*This does not mean that their hearing was really normal, but only that no information to the contrary was recorded by the enumerator on the schedule.

Area	States
East	Massachusetts, New Jersey, New York, Pennsylvania.
Central	Illinois, Michigan, Minnesota, Missouri, Ohio.
South	Alabama, Georgia, Louisiana, Texas, Virginia.
West	California, Oregon, Utah, Washington.

Sampling procedures were employed in 31 cities in which the population exceeded 100,000 inhabitants. The sampling ratios varied in these cities from 1:4 to 1:38. In a few instances, cities with less than 100,000 population were sampled, with the ratio 1:2. Other cities and towns were enumerated 100 per cent.

TABLE I.

Distribution of Population Enumerated During the National Health Survey (1935-1936), According to Geographic Area and Size of City in Four Broad Groups.

Size of City	Cities		Population in Health Survey Sample					
			All Areas					
United States Census of 1930	Number	Per Cent	Number	Per Cent	East	South	Central	West
500,000 and over..	10	12.1	1,077,449	43.0	584,120	.....	416,622	76,707
100,000 to 500,000	21	25.3	767,193	30.7	172,417	251,540	205,169	138,607
25,000 to 100,000	10	12.0	350,605	14.0	69,380	132,173	125,357	23,695
Under 25,000.....	42	50.6	307,144	12.3	87,503	71,928	77,128	70,585
Totals	83	100.0	2,502,391	100.0	913,420	455,641	824,276	309,054

The geographic distribution according to area and size of city of the Health Survey population sample is given in Table I. There were altogether 2,502,391 persons, of whom 1,201,992 (48 per cent) were male, and 1,300,399 (52 per cent) were female. These persons were members of 703,092 households. There were 744,205 informants in these households who gave schedule information to enumerators. Among the informants there were 170,594 males and 573,611 females. The schedule information was given, therefore, in 77 per cent of the cases by females, most of whom were housewives. There were on the average 3.36 household members, including the informant, for each informant. Thus, in 70 per cent of the cases the enumerators' returns on the schedules represent information about persons other than the one giving the information. This fact is important, especially in respect to

reports on impaired hearing, since it was found that at less severe levels of hearing loss individuals recognize their own defects of hearing more readily than those of other people.

The gross size of the Health Survey population sample (observed in 1936) is equivalent to 3.7 per cent of the 1930 urban population of the United States. It is impossible to state whether the canvassed population was representative, in a rigorous sense, of the whole urban population in 1936 and certainly it is not a random selection from the entire population. The Health Survey population may be described quite appropriately as a representative cross-section or stratified sample of urban United States in 1936.

SAMPLING WITHIN CITIES.

On the basis of administrative and various other considerations, a quota of schedules was assigned to each city. For each city that was to be sampled, a table was set up showing enumeration districts which were employed by the Bureau of the Census in 1930, and the population size of each district. A sufficient number of these enumeration districts was selected at random from the table to yield a total population to correspond with the total number of schedules assigned to that city. From this procedure it is considered that a relatively unbiased sampling of enumeration districts was achieved. The map shown in Fig. 1 illustrates a typical scatteration of enumeration districts as drawn in the manner described for the purposes of the National Health Survey in Atlanta.

There are four valuable characteristics of the information collected in this particular survey which provide a basis for studying certain epidemiological aspects of impaired hearing in the general population. First, the wide range of socio-economic facts about the persons included in the census has proved to be quite pertinent because the cross-tabulations reveal that impaired hearing varies, both in prevalence and kind, with several of these factors. Unless considerable attention is given to evaluating the population sample in respect to such factors as age, sex, employment status, occupation, family income and the relative amount of sickness in the various subgroupings, an appraisal of the generalized incidence estimates cannot be accomplished. Secondly, in addition to the classification of hearing impairments in terms

of the above definitions, other information for each deafness case reported was obtained, such as age at onset of deafness, whether the deafness is progressive or stationary, and whether the deafness interferes with the person's occupation or is disabling in the sense of an employment hazard. Thirdly, there is apparently no disturbing bias in the population sample in respect to the rather broadly classified socio-economic characteristics of the families. And fourthly, the

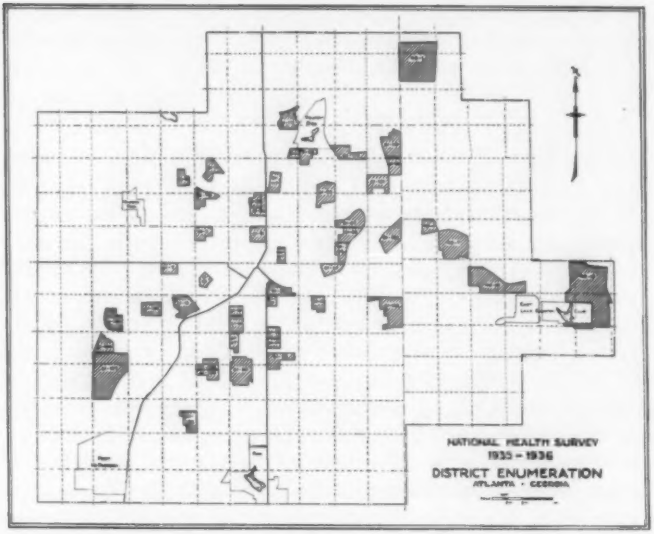

Fig. 1. Shaded areas show boundaries of enumeration districts in which all households were canvassed during National Health Survey, Atlanta.

data tabulated from the enumerators' returns on the schedules can be evaluated in terms of hearing loss measurements which were obtained during the supplementary clinical investigation, at which both audiometric and otologic examinations were carried out on persons who had been included in the household census.

THE CLINICAL INVESTIGATION.

The schedules resulting from the household census were shipped to a central statistical unit in Detroit during the

progress of enumeration in the various cities. After the completion of the Health Survey, a random sample of these schedules was drawn from the returns for 12 of the 83 cities. For each of these 12 cities all schedules which contained entries for deafness, any stage, were segregated. These are referred to as "case" schedules. A considerably larger random sample of the schedules on which no entries for deafness had been made was drawn. These are "no-case" schedules. Identifying and other information transcribed from these schedules was used as a basis for investigating impaired hearing in relation to the information which had been given originally on the schedules to the enumerators.

Seventeen temporary clinics were set up in the following 12 cities:

Alabama: Birmingham, Montgomery.

Georgia: Atlanta, Brunswick.

Louisiana: New Orleans.

Michigan: Detroit.

Minnesota: St. Paul.

New York: Brooklyn.

Ohio: Cleveland.

Texas: Amarillo, Dallas, Houston.

All persons in each household that was included in the drawn sample of both the "case" and "no-case" schedules were invited to these clinics for an ear, nose and throat examination and audiometric test. Practicing physicians and acoustical engineers were appointed by the United States Public Health Service for temporary duty in expediting the work of these clinics. These professional persons were responsible directly for collecting the scientific data. Clerical and nontechnical assistance was obtained through co-operation of the Works Progress Administration.

Ear, nose and throat examinations and medical histories were obtained for each subject who attended the clinic. Attendance was voluntary and excellent co-operation by the subjects was universal. Auditory acuity measurements were obtained on eight tones of the Western Electric 2A audiom-

eter for both ears by air conduction, and on six tones by bone conduction. All hearing tests were carried out in sound-insulated booths of uniform construction. Tests on the efficiency of these booths indicate that they attenuate sounds in the external environment to the extent of 28 to 34 dcb. The steady noise level in the booths varied from less than 25 dcb. to approximately 42 dcb.*

Careful attention was given to securing reliable auditory acuity measurements. For more than 50 per cent of 9,324 subjects studied at the clinics, two complete sets of audiograms were obtained on both ears by air and bone conduction for both the ascending and descending approach to threshold. For the remainder of the subjects, one complete set of audiograms was obtained for the ascending and descending approach to threshold on both ears for eight tones by air conduction, as well as for six tones by bone conduction. Adequate time was allowed for the audiometric tests and several recheck measurements were obtained for each threshold determination, depending upon the consistency and co-operation of the subjects.

When the physician or his assistant obtained information from a subject at the clinic for the medical history form, the same questions which were asked during the household census were repeated. There were some slight differences in procedure in asking questions about the person's condition of hearing, and there were marked differences between other aspects of the clinical interviewing situation and that which existed during the household census. These two factors must be taken into account in estimating deafness in the population from the materials of this study.

At the clinical interview, normal hearing was defined as "no noticeable difficulty" and each person was asked whether he had ever experienced difficulty with his hearing. Persons were classified as having a clinical history of normal hearing only when the answer to this question was "no." Basic definitions for stages of deafness were otherwise the same as those employed during the household census; however, many notations appeared on history forms indicating noticeable

*Average levels as measured on the "Ear 40, 70 and Flat" scales of the Type RA-224-C sound level meter, manufactured by Electrical Research Products, Inc.

defects of hearing not regarded as severe as stage 1. Also, there were some cases in which persons claimed to have recovered from previously noticeable difficulty with hearing. For purposes of clinical history classifications, these latter two groups of individuals were designated as stage 1 deafness.

The clinical history classifications of hearing difficulty are based on information which was obtained in each case, of course, directly from the individual being interviewed. The physician ascertained from direct observation whether the subject could hear conversation at close range or whether he could hear loudly shouted words close to his ears. The opportunity to make these observations at the time the history was being recorded enabled the physician to classify the subject's hearing difficulty much more accurately in terms of the definitions of the various stages than could possibly have been the case during the household census. The clinical history classifications were made independently of the audiometric examinations, so that the doctor's judgment was not influenced by the nature of the subject's audiograms.

It is recalled that 70 per cent of the enumerators' classifications in respect to impaired hearing were secured for persons other than the one giving the information. For convenience of terminology, it is considered that the reports by enumerators may be meaningfully described as "social" history of impaired hearing in contrast with the kind of situation in which the "clinical" history was obtained by a physician. The results of the audiometric tests for both air and bone conduction were tabulated, first, according to the social history classifications of impaired hearing; and, secondly, according to the clinical history of normal or impaired hearing. The average hearing losses for the subjects in each of these groups were computed. Audiograms based on these averages are shown in Fig. 2.

HEARING LOSSES ACCORDING TO SOCIAL HISTORY OF IMPAIRED HEARING.

A good audiometric index to the ability of an individual to hear speech sounds is obtained by taking the average of hearing losses by air conduction on 1,024 and 2,048 cycles. The

average losses on these two tones is 7 dcb. for persons not reported on the Health Survey as having impaired hearing. Persons are reported as having stage 1 deafness or worse when their average hearing loss is 47 dcb. or more on 1,024 and 2,048 cycles. Stage 2 deafness, according to the social history, is at a level of 55 dcb.; stage 3 deafness is at 68 dcb.; and total deafness for speech is at 89 dcb. It is observed also, from the nature of the bone conduction audiograms shown in Fig. 2, that there is marked hearing loss by bone conduction for all stages of deafness reported on the Health Survey.

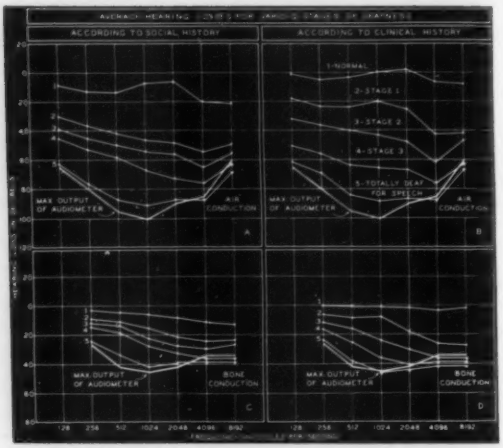

Fig. 2. Average levels of hearing loss for various stages of impaired hearing, according to social history (household census) and clinical history (personal statement to physician).

From the character of these losses it is inferred that hearing impairments among these people are predominantly nerve deafness. This finding is at variance with a commonly held view that most deafness is essentially conductive loss.

HEARING LOSSES ACCORDING TO CLINICAL HISTORY OF IMPAIRED HEARING.

A comparison of the average air-conduction audiograms according to social history (see Fig. 2A) with those for clinical history of hearing difficulty (see Fig. 2B) reveals certain

striking differences. Normal hearing according to clinical history is at an average level of —0.3 dcb. on 1,024 and 2,048 cycles, which is 7.3 dcb. less than the averages for social history of normal hearing. This figure is significant because the majority of ears in both groups exhibit normal hearing in all respects. Consequently, a rather large number of cases with impaired hearing must be introduced into the distribution of cases for the "socially" normal group in order to influence the arithmetic mean to the extent shown.

A comparison of the average hearing loss levels for stage 1 deafness, according to the two types of history, supplies a partial explanation for a considerable proportion of the differences between averages for the two normal hearing groups. According to the clinical history, stage 1 deafness is equivalent to an average hearing loss of 24 dcb. on 1,024 and 2,048 cycles, as compared with 47 dcb. for social history of stage 1 deafness. It is evident that stage 1 deafness, according to clinical history, is missed almost entirely in the household census technique of securing this type of information and that, on the average, impaired hearing is not recognized socially until the individual has attained an impairment sufficient to interfere with understanding direct conversation. When the individual himself gives the information, as at the clinic, impaired hearing is recognized by the person having an impairment when hearing loss has occurred to the extent of 20 to 25 dcb. for 1,024 and 2,048 cycles.

The differences between average hearing losses that represent the various conditions of hearing according to social and clinical histories provide a basis for estimating the distribution of impaired hearing according to clinical history norms for the general population. On the other hand, the average hearing losses given for the social history groups alone provide a direct basis for interpreting in terms of amount of hearing loss the prevalence rates for various stages of deafness as obtained directly in the household census. It is clear that all of the data on impaired hearing as obtained during the census represent cases of hard-of-hearing persons; that is, ones having considerable difficulty even with direct conversation. The household census data are considered to be in error to the extent that: *a.* stage 1 deafness, according to clinical history criteria, was missed almost entirely (not

reported); and *b*. there is an unknown number of persons with severely impaired hearing who were not reported.

It is of interest to interpret the average air-conduction audiograms shown in Fig. 2 for the various social and clinical histories of auditory impairment in terms of hearing loss levels on the Western Electric 4A audiometer, since extensive statistics on hearing defects among children of public school age, based on results from tests given with the 4A audiometer, have become available in recent years. In 1936, Ciocci[15] reported data showing the relationship between hearing loss, as measured by the 4A audiometer, and the thresh-

TABLE II.

Relationship Between Hearing Losses as Measured by Western Electric 4A and 2A Audiometers.

Hearing Loss in Decibels on 4A Audiometer	Average Hearing Loss in Decibels for 1,024 and 2,048 Cycles on 2A Audiometer
—3	6.5
0	9.1
3	9.5
6	10.2
9	12.4
12	15.2
15	25.3
18	29.7
21	41.3
24	40.0
27	47.5
30+	52.4

old of acuity for pure tones as measured by the 2A audiometer. The measurements made with the 2A audiometer were grouped according to the better score made by each ear in either of two successive 4A tests. These measurements were carried out on 1,400 school children in Washington, D. C., during the school year 1931-1932. The summary in Table II shows the average hearing loss on 1,024 and 2,048 cycles (independent variable) at each hearing loss level of the 4A audiometer (dependent variable).

Since later in this discussion rates of prevalence for impaired hearing as determined from the data collected during the household census will be reviewed, it is of chief interest to compare the average hearing losses on 1,024 and 2,048 cycles at the various levels of hearing loss on the 4A audi-

ometer (see Table II) with the average hearing loss levels on 1,024 and 2,048 cycles for the various *social* histories of hearing impairment (see Fig. 2A). It is observed that cases of stage 1 deafness, as reported on the Health Survey, represent *on the average* hearing impairments equivalent to 30 SU or more loss on the 4A audiometer. This fact should be borne in mind when interpretation is made of the rates of prevalence derived from the census data.

TABLE III.

Prevalence of Severely Impaired Hearing of Various Stages, Showing Number of Persons per Case of Deafness in the Health Survey Population (1935-1936).

Social History of Hearing Difficulty (Household Census)	Chronological Age in Years									
	All ages	Under 5	5-14	15-24	25-34	35-44	45-54	55-64	65-74	75 & over
	Male									
All stages of deafness	78	2,041	339	285	210	103	67	34	14	6
Partial deafness, stage 1	152	9,091	719	541	417	189	123	64	26	13
Partial deafness, stage 2	292	.....	1,538	1,563	962	410	261	123	49	19
Partial deafness, stage 3	503	.....	3,125	3,333	1,754	833	469	232	81	27
Totally deaf for speech	2,326	11,111	5,882	3,846	3,571	3,333	2,564	1,429	541	141
	Female									
All stages of deafness	85	2,326	442	341	200	108	65	38	18	7
Partial deafness, stage 1	181	8,333	962	658	385	211	136	81	40	18
Partial deafness, stage 2	294	.....	2,273	1,818	794	391	209	127	61	24
Partial deafness, stage 3	508	.....	3,704	3,125	1,754	800	418	216	101	34
Totally deaf for speech	2,000	20,000	7,143	6,667	5,882	3,030	1,923	926	402	167

RATES OF PREVALENCE FOR VARIOUS STAGES OF IMPAIRED HEARING.

Rates of prevalence, which state the amount of a given characteristic existing at any one time in a population, may be expressed in terms of the number of persons per case of the characteristic; or in terms of the number of cases of the characteristic in ratio to some fixed number of the population, such as 100 or 1,000. The data given in Table III express prevalence of impaired hearing in terms of the num-

ber of persons under each classification per case of impaired hearing of the degree specified. The ratios were computed for the Health Survey population as observed, without any standardization. For the Health Survey population as a whole, one out of every 78 males and one out of every 85 females had an impairment of hearing to the extent of 47 dcb. or worse (average loss on 1,024 and 2,048 cycles). Among children of grammar school age (5 to 14 years), one out of every 339 males and one out of every 442 females have stage 1 deafness or worse. These are mostly hard-of-hearing children who require training in special schools, and few of them are

Table IV.

Prevalence and Annual Incidence Rates for Impaired Hearing (Any Stage) According to Age and Sex. Calculations are based on National Health Survey Experience of 1935 and 1936.

Age Period (Years)	PREVALENCE Deafness Cases (Any Stage)* Per 1,000 Population		INCIDENCE Average Annual Rate† of New Cases Per 100,000 Population	
	Male	Female	Male	Female
Under 5	0.49	0.43	.....	.....
5 to 14	2.95	2.26	33	24
15 to 24	3.51	2.93	6	7
25 to 34	4.76	4.99	13	21
35 to 44	9.70	9.28	49	43
45 to 54	14.93	15.45	52	62
55 to 64	29.27	26.43	143	110
65 to 74	73.64	54.68	444	283
75 and over	175.08	135.95	1,014	813

*Equivalent to an average hearing loss of 47 dcb. or more or 1,024 and 2,048 cycles.

†New cases per year at each single-year age throughout specified age period.

able to keep up with normal hearing children in the regular public schools. There are marked differences between the prevalence rates of severely impaired hearing among successively older persons and in similar degree for both sexes. At ages 35 to 44 years, one out of every 103 males and one out of every 108 females have a hearing loss of 47 dcb. or worse. In the age period 65 to 74 years, one out of every 14 males and one out of every 18 females are are hard-of-hearing in the sense of the above criteria.

Prevalence rates given in Table IV are stated in terms of the number of cases (stage 1 or worse) per 1,000 population,

according to age and sex. These rates are employed in plotting the trend curves shown in Fig. 3.

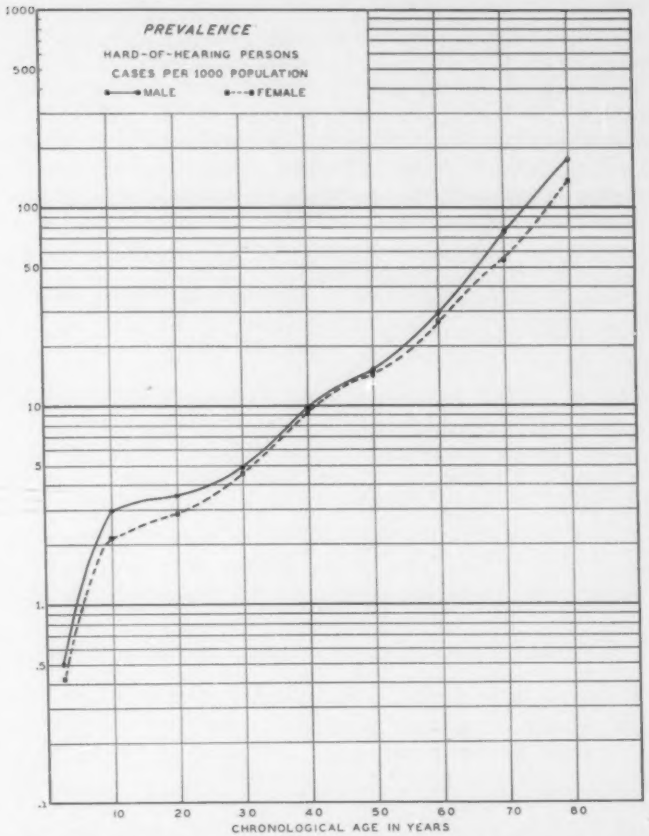

Fig. 3. Trend curves, based on returns from National Health Survey household census, showing prevalence of impaired hearing (stage 1 or worse) among males and females at various ages.

There is consistently higher prevalence of severely impaired hearing among males in all age groups, excepting the period 25 to 34 years, during which there is a slightly but not significantly higher rate for females. The two curves exhibit essentially the same trend with age for both sexes. It should

be noted that these curves are plotted on a semilogarithmic scale. If new cases of deafness of the severity under consideration occurred at a uniform rate during all age periods, these curves would plot as straight lines. It is observed, however, that there are several age periods in which the curves slope steeply upward (5 to 10 years, 30 to 40 years, and over 55 years), and during other periods they level off more horizontally (10 to 30 years and 40 to 55 years). These variations are of significant magnitude, and they indicate age periods during which combined factors produce severely impaired hearing at relatively higher rates, as will be discussed below under trends of incidence.

It is of considerable interest to note that for the age period 5 to 14 years, the prevalence of impaired hearing as obtained on the Health Survey household census is 2.95 per 1,000 for males and 2.26 for females. As shown above, these cases are equivalent to 30 SU or more loss on the 4A audiometer. There is rather close agreement between these rates and those reported from school surveys for this degree of hearing loss on the 4A audiometer. Thus, Ciocco[15] gives the figure 2.3 cases per 1,000 ears tested, both sexes, based on 27,964 ears; Fowler[25] found 2.4 cases per 1,000, better ears for both sexes; Fowler and Fletcher[33] give the rates for tests conducted in four New York public schools varying from 1.0 to 4.0 per 1,000, averaging about 2.6 per 1,000 for all schools.

ANNUAL INCIDENCE RATES OF NEW DEAFNESS CASES.

Deafness is a chronic impairment, typically progressive. There seldom is recovery of good hearing once an individual has become hard-of-hearing. Crowe and Guild[19] reported a case in which good hearing was restored through X-ray therapy on a boy, age 14 years, showing initially losses on all tones ranging from 30 to 60 dcb. by air conduction. This clinical demonstration is encouraging indeed, but spontaneous recovery in similar degree is extremely rare. Most frequently, hearing impairments show progressively greater losses for high tones with advancing age; infrequently, impairments remain fairly constant over a period of years. Aural risks to which individuals are exposed predominate in childhood, young adult and middle age periods of life. The specific characteristics of risks that are most prominent vary

at different periods of life, although during all age periods risks *per se* are present. The *pathogenicity* of aural risks, that is, their ability to produce pathologic changes in the auditory mechanism, likewise varies, even though exposure occurs. And, finally, susceptibility of the individual to pathogenic exposures varies with age, sex and a multiplicity of other factors.

Despite the complex mode of variation in these numerous factors which contribute to the production of impaired hearing, they add up cumulatively in the lifetime of individuals. Consequently, rates of prevalence for impaired hearing, all types and all degrees, must gain in magnitude among successively older groups, providing that those who experience this impairment do not suffer a higher mortality rate than persons not so impaired. The prevalence rates would show cumulative gains anyway, unless differential mortality rates equaled or exceeded the increments in impaired hearing cases. The rates of prevalence given in Table IV and illustrated by the trend curves in Fig. 3 exhibit this cumulative increase in magnitude for successively older groups of persons; and, as pointed out above, the nature of the curves indicates that the gain in magnitude per decade is not a constant ratio of the amount of deafness existing in the next preceding decade. Consequently, it is apparent that aural morbidity has periods of acceleration and retardation in relation to age, which may be revealed by calculation of incidence rates for various age periods.

Rate of incidence measures the change in amount of a given characteristic in an observed population over a specified period of time. It measures rate of change in rate of prevalence; or, an incidence curve is the first derivative of the curve of prevalence. Annual incidence rates can be derived from the prevalence rates given in Table IV by making two assumptions: *a.* that persons in each decade age group of the Health Survey population are representative of persons in the same age groups of the general population *in respect to* the prevalence of impaired hearing among them; *b.* that mortality is indifferently related to acquisition of impaired hearing, *or* that whatever relation exists between mortality and impaired hearing, it is a *constant factor* at all ages. With these assumptions in mind as possible limitations

on subsequent conclusions, we may derive incidence rates from the prevalence rates.

The prevalence rates given in Table IV represent amounts of impaired hearing per 1,000 population in decade age groups with the exception of the youngest group, which is one-half decade. It is necessary to assume a representative age for each group; and so the median value is chosen. Thus, for the group "under 5 years" the value is 2.5 years; for the group "5 to 14 years" the value is 10 years; etc., for the other groups. The estimated median for the group "75 years and over" is 80 years. The difference between the prevalence rates (cases per 1,000 population) for two successive age groups when divided by the difference (in years) between the median values of the two age groups yields a quotient which is the average annual increment (or incidence) of new cases per year per 1,000 population through the period denoted by the median age values. This quotient, when multiplied by 100, expresses the average annual incidence in cases per 100,000 population, as given in Table IV under incidence. The divisor is 10 years for all pairs of periods except the first two, in which case it is 7.5 years.

These derived rates are more instructive than the prevalence rates *per se*, since they indicate directly the periods of relatively greater activity in production of new cases of impaired hearing in the degree estimated from the results of audiometric tests shown above in Fig. 2. The age period from 2.5 to 10 years is relatively more serious than the whole period from 10 to 30 years. During this early childhood period, new cases of impaired hearing to the extent of 47 dcb. or more average loss on 1,024 and 2,048 cycles appear annually on the average at the rates of 33 cases per 100,000 males and 24 cases per 100,000 females. During the period 10 to 20 years the rates fall off to six and seven, for males and females, respectively, and rise to 13 for males and 21 for females during the period 20 to 30 years. The rate of incidence rises rapidly for males and females during the period 30 to 40 years, remains practically the same for males on through the period 40 to 50 years but continues to rise rapidly for females during this period. After 50 years the rate of incidence rises again for males, attaining the value 444 for the period 60 to 70 years, and 1,014 at ages over 70 years.

Among females the gain in rate of incidence during the period 60 to 70 years is only about half that experienced by males, the value being 283. At ages over 70 years, females attain the rate of 813. Although this is smaller than the rate for males of the same age range, it represents a relatively greater gain over the preceding decade than that for males.

RELATIONSHIP BETWEEN PREVALENCE OF DEAFNESS AND FAMILY INCOME.

Impaired hearing as severe as that represented by stage 1 deafness or worse in the Health Survey census is a serious personal, social and occupational disability. Such impairments likewise represent functional degeneration or damage to an important sense organ, arising most often from attack by both chronic diseases and acute illnesses, aside from destructive influences associated with the nature of one's physical environment and habits of life. General systemic deficiencies, occasioned by malnutrition, toxic conditions and various other liabilities upon general health, may be expected to exert an influence on the frequency and severity of aural morbidity. In a direct way this complex host is related to material standards of living, which in turn are determined to a large extent by economic status or total financial assets of the family. It is of interest in the present epidemiological approach to a study of impaired hearing to inquire concerning possible relationships between income and the rate of prevalence for severely impaired hearing; but in so doing it is well to bear in mind that the variables being compared are infinitely complex, and only very general observations can be expected from such comparisons.

Prevalence of impaired hearing (stage 1 and worse) in relation to total family income as observed during the National Health Survey is given in Table V, according to sex, four broad age groups and three broad income levels. The rates are expressed in relative values, the base reference of 100 being used for persons in families having a total income of $3,000 and over. Presentation of the values in this manner facilitates evaluating the difference between the various groups associated with income alone.

It is noted that for males and females of all ages, income exerts a relatively greater influence on the prevalence of

impaired hearing among males than among females, the rate for males in the lowest income families being 74 per cent higher than that for the highest income group; whereas for females, the difference is 38 per cent. Curiously, there is a reversal in the relative difference with age for the two sexes. The relative differences between rates of prevalence according to income *increase* with age among males, but they *diminish* with age among females. At ages under 25 years the relative difference between rates of prevalence for lowest and highest income groups are greater among females than among males. At all ages over 25 years, this relative difference is greater

TABLE V.

Relative Prevalence of Impaired Hearing (Any Stage) According to Family Income, Age and Sex. National Health Survey (1935-1936).

Family Income	Relative Prevalence Rate				
	Age Range in Years				
	All Ages	Under 25	25-44	45-64	65 and Over
	Male				
Relief and under $1,000	174	150	185	210	190
$1,000 to $2,999	102	101	120	119	124
$3,000 and over	100	100	100	100	100
	Female				
Relief and under $1,000	138	175	133	135	124
$1,000 to $2,999	110	125	103	103	102
$3,000 and over	100	100	100	100	100

among males. The magnitude of these differences are significant in all instances where comparisons are made between the rates for the lowest and highest income groups. This differential sex trend may reflect exposure to more severe aural risks experienced by males in connection with their occupations on the assumptions that males over 25 years of age *a.* are wage earners more often than females; and *b.* that male wage earners in low income classes are subject to greater aural risks in industry and other hazardous occupations than females, who become housewives in the majority of cases at ages over 25 years. Another factor which might be supposed to contribute to this trend is that males who are hard-of-hearing tend to gravitate to lower income groups because their aural handicap is an occupational liability and, since they are the wage earners in the majority of families, the income classification reflects their reduced wages. Hence,

the lower income groups would tend to acquire relatively more hard-of-hearing males during the active employment period 25 to 64 years than higher income groups because of this *shift in economic status*. A similar trend would not occur among females for the same reasons because *a*. the wages of relatively fewer females enter into the determination of the economic groups as tabulated; and *b*. the acquisition of impaired hearing by housewives would not affect their economic status as in the case of wage earners. In reference to the reversal of this trend for the two sexes at ages under 25 years, there seems to be no immediately apparent explanation. As a matter of fact, the above explanation concerning the differential trend at ages over 25 years is entirely supposition, reasonable though it seems; but no direct evidence is available in the present data to support the assumptions. It can be said with assurance, however, that these differential trends in relation to family income are significant, despite the lack of satisfactory explanation.

RELATIONSHIP BETWEEN OCCUPATION, EMPLOYMENT STATUS AND PREVALENCE OF IMPAIRED HEARING.

Tabulations showing the relationship between prevalence of impaired hearing (stage 1 or worse, household census) and occupation are presented in Table VI. In order that several different important trends may be observed, the data are broken down in detail by decade age groups, sex and whether or not the individuals were employed (autumn of 1935, winter and spring of 1936). In the preparation of this table, the following groups were excluded: persons attending school; persons "at home," that is, retired or not seeking work for other reasons; persons who had been in an institution for the care of disease or impairment during the entire 12 months immediately preceding the enumerator's visit; all persons under 15 years of age; all persons of unknown age, unknown occupational class or unknown employment status. Housewives were included as a control in the comparison of rates for various female groups.

Among employed males, the highest prevalence of impaired hearing occurs among skilled and semiskilled workers in industry; the second highest rates are found among other semiskilled and unskilled workers; and the lowest prevalence

occurs among professional, business and clerical persons. Similar variations are noted for employed females. Equivalent relative differences between the rates for the unemployed,

TABLE VI.

Prevalence of Impaired Hearing (Any Stage) According to Occupational Class, Employment Status, Age and Sex. National Health Survey (1935-1936).

Occupational Class and Employment Status	Prevalence Rate: Cases per 1,000 Population Age Range in Years					
	15-24	25-34	35-44	45-54	55-64	65 & Over
	Male					
Employed all	2.97	4.12	8.46	12.75	22.46	57.18
Professional, business	2.57	3.48	7.39	10.11	19.90	51.09
Clerks, salesmen	2.53	2.89	7.59	11.48	17.41	50.83
Skilled workers	4.49	5.71	10.74	15.63	26.03	63.02
Semiskilled workers in industry	2.32	5.64	10.63	16.79	31.41	63.12
Other semiskilled	2.45	3.06	7.25	10.49	17.66	49.42
Unskilled	3.29	5.26	7.12	13.24	24.35	59.26
Farmers, farm laborers, servants	3.93	5.55	6.22	10.98	24.33	73.72
Unemployed, all	4.24	7.47	15.74	22.26	42.68	101.29
Professional, business	3.79	6.80	17.03	19.98	34.28	79.53
Clerks, salesmen	3.60	7.18	14.14	23.35	32.99	87.43
Skilled workers	4.96	9.05	17.25	24.97	49.38	111.16
Semiskilled workers in industry	6.43	8.04	17.25	24.54	44.30	113.36
Other semiskilled	4.62	5.74	14.56	21.21	41.78	81.13
Unskilled	3.72	5.96	12.68	16.25	32.10	93.87
Farmers, farm laborers, servants	3.98	9.24	18.38	25.69	58.23	117.27
	Female					
Employed, all	1.86	2.99	6.22	10.52	19.63	49.90
Professional, business	1.41	2.30	4.85	9.14	16.32	49.37
Clerks, salesladies	1.34	1.98	6.06	5.94	16.98	62.12
Semiskilled workers in industry	2.57	5.80	9.15	17.21	35.11	56.11
Other semiskilled	2.55	3.72	6.35	15.18	23.98	58.51
Farmers, farm laborers, servants	2.12	3.11	5.79	9.48	19.00	39.43
Unemployed, all	3.35	6.34	11.56	18.71	35.50	68.79
Professional, business	2.38	8.32	21.71	30.89	48.17	71.43
Clerks, salesladies	2.13	6.00	10.67	22.83	30.81	57.14
Semiskilled workers in industry	4.56	8.54	13.97	28.49	45.87	61.77
Other semiskilled	4.09	7.08	13.03	38.00	52.38	87.81
Farmers, farm laborers, servants	3.45	5.02	7.97	11.42	39.53	68.18
Housewives	3.76	5.69	9.96	16.24	26.29	75.30

classified according to their usual occupation, is observed. The prevalence of hearing impairments among housewives is seen to be considerably higher at all ages than among *employed* females, a trend which runs counter to usual opinion; on the other hand, unemployed females who are usually employed and who, at the time of the census, were *seeking work* show on the whole higher prevalence of impaired hearing than housewives. It should be mentioned here that a separate critical evaluation of the rôle played by the informant in providing information about household members resulted in the conclusion that informants reported data concerning themselves better than they reported data on other household members. This is true in respect to prevalence of impaired hearing for both males and females. For example, there is a higher prevalence of impaired hearing among male informants than among male noninformants; likewise, there is higher prevalence of impaired hearing among female informants than among female noninformants. These are general trends and probably enter into the differences between rates for housewives as shown in Table VI and the other occupational groups of females, especially since the majority of females actually are housewives and served as informants. This trend does not influence the differential rates for males, since only a small percentage of them served as informants during the census.

The most conspicuous and probably the most significant trend revealed in Table VI is the excess prevalence of impaired hearing among the unemployed in each occupational class. Estimates concerning the significance of these differences were computed for the whole table by setting up a 2x2 fold dichotomy for each cell from the original count of cases, showing number of persons employed and unemployed, according to whether they did or did not have impaired hearing as revealed by the census returns. This procedure yielded 42 paired dichotomies for males and 30 for females. Housewives, of course, did not enter into these tests. Application of the Chi-square test of independence to these individual sets of paired dichotomies (one for each age, occupational and sex group) showed that in no case could the hypothesis of independence be maintained. The relation is definite in these tabulations that prevalence of impaired hearing and whether the person is employed show dependence.

Such a trend might be supposed to have arisen from the complex of factors which in general yield higher sickness rates among persons on relief or with low economic status; *i.e.*, inadequate and inappropriate food, insufficient medical care, increased mental anxiety and resulting degeneration of personal habits, and other similar factors. This population was observed, however, during the immediate sequel of the depression, and most of the unemployed population had acquired that status rather recently in relation to the time of the household census. It is doubtful, therefore, that factors related directly to economic status would produce this higher prevalence of impaired hearing among the unemployed in such a brief period of time. It appears more reasonable to assume that the significantly higher prevalence of impaired hearing among the unemployed reflects selection by employers in favor of employees who were not hard-of-hearing at the time when large-scale layoffs occurred. Since large numbers of persons were being released from their regular employment due to industrial and business failures during the period from 1930 to 1935, it is common sense expectation that the less capable (mentally) and the physically handicapped would be discharged before others. The growing unemployed population during this period would naturally accumulate a disproportionately large composition of so-called less fit, incompetent and physically handicapped persons, at least as so viewed by some and possibly most employers.

This interpretation is substantiated partially by the observation that ratios between prevalence rates for impaired hearing among the employed and unemployed vary considerably among the different occupational classes. For example, such ratios are much larger for persons in professional, business, and clerical or sales groups than for skilled and semi-skilled workers in industry. Good hearing is a more important consideration in the former than in the latter occupations. Moreover, if factors associated with economic status were responsible for the higher prevalence of impaired hearing among the unemployed, one would expect a differentially greater prevalence among the occupations which in general yield lower incomes, since persons in these occupations would have been exposed to the composite risks associated with low income for a much longer period of time and, hence, cumu-

lative effects from such exposure would be more pronounced among them, as reflected by higher prevalence of impaired hearing. As pointed out above, just the reverse of this actually happens; that is, there is a greater relative difference between prevalence rates for impaired hearing among the employed and unemployed in occupations which usually yield larger incomes than between those in occupations which usually yield smaller incomes.

Regardless of whether or not persons were employed at the time of the census, impaired hearing occurs with considerably higher prevalence among skilled and semiskilled workers in industry than in any of the other occupational classes. This is true for both males and females. It is apparent, therefore, that aural risks peculiar to working conditions in industry produce severely impaired hearing in excess of that arising either from other occupations or from composite risks associated with lower (economic) standard of living. The data show also that severely impaired hearing is a greater employment hazard in some occupations (professional and business persons) than in others (skilled and semiskilled workers in industry, as well as unskilled workers generally). In addition to the fact that the degree of employment hazard varies with kind of occupation, the results of this study provide evidence that severely impaired hearing is an employment hazard in all occupations as classified for purposes of this analysis.

RELATIONSHIP BETWEEN DEGREE OF DEAFNESS AND PREVALENCE OF UNEMPLOYMENT.

In the preceding section it was shown conclusively that a relation of dependence, significant in degree both practically and "statistically," exists between the two dichotomies, "employed or unemployed" and "impaired hearing or not impaired hearing." The relation is significant for either males or females in each of seven occupational groups of males and five occupational groups of females, for each of six age decades. Despite the demonstration that such a relation of dependence exists in the data as distributed in Table VI, a direct relation between impaired hearing and unemployment is not established. Both corroborative and more convincing evidence of dependence could be revealed, if it were

possible to show varying degrees of unemployment prevalence correlated with varying degrees of hearing impairment. The tabulation presented in Table VII was prepared to discover whether such a correlation would be exhibited.

TABLE VII.

Relationship Between Unemployment Prevalence Rates, Usual Occupation and Condition of Hearing.

Usual Occupation	Unemployment Prevalence Rates in Per Cent			Ratios Between Unemployment Prevalence Rates	
	Good Hearing a	Moderate Deafness b	Severe Deafness c	b/a	c/a
ALL occupations	23.0	36.9	45.0	1.60	1.96
Professional persons, business managers and officials	9.3	18.3	29.2	1.97	3.14
Clerks, salesmen and kindred workers	14.4	25.4	34.1	1.76	2.36
Foremen and skilled workers	26.8	44.7	50.1	1.67	1.87
Semiskilled workers.. in manufacturing	22.2	38.0	41.7	1.72	1.88
Other semiskilled workers	20.2	30.8	38.7	1.52	1.92
Unskilled workers, except farm laborers and servants	41.0	55.8	58.1	1.36	1.42
Farmers, farm laborers, servants	41.4	47.7	56.1	1.15	1.35

The table shows unemployment prevalence rates in per cent for seven occupational classes (males and females combined, all ages), according to three classifications of hearing condition: *a*. those with a social history of normal hearing; *b*. those with a social history of either stage 1 or stage 2 deafness; and *c*. those with a social history of stage 3 or total deafness for speech. In terms of hearing ability as determined from audiometric examinations, those in group *a* (good hearing) have *on the average* hearing loss of 7 dcb. for 1,024 and 2,048 cycles; those in group *b* (moderate deafness) average 51 dcb.; and those in group *c* (severe deafness) average 76 dcb. The hearing losses of these three major groups are separated to such an extent as to constitute marked differences in ability to hear speech sounds in any situation usually encountered in everyday life or under laboratory conditions.

The prevalence rates in column *a* of Table VII, for persons with good hearing, may be construed as reflecting the dif-

ferential unemployment condition for various occupational groups in 1935 and 1936. It is observed that professional and business persons in general suffered less unemployment (9.3 per cent), and that rates of prevalence are increasingly greater from upper to lower groups in this socioeconomic scale of occupational classes, attaining a high value of 41 per cent unemployed among unskilled workers, farmers, farm laborers and servants.

The ratios given in the last two columns on the right side of the table indicate the influence of moderate (group *b*) or severe (group *c*) deafness on prevalence of unemployment, relative to the rate given in each occupational class for persons with good hearing. Considering all occupational classes, we observe that unemployment prevalence is 60 per cent higher among persons with moderate deafness, and 98 per cent higher among those with severe deafness, as compared with the rate of unemployment among those with good hearing.

More striking are the variations in the relative influence from degree of hearing impairment on unemployment prevalence in the different occupational classes. Thus, among professional and business persons, unemployment is 97 per cent higher among those with moderate deafness, and 214 per cent higher among those with severe deafness, as compared with the group having good hearing. The least influence on unemployment associated with impaired hearing is found among farmers, farm laborers and servants. In this group, unemployment prevalence is only 15 per cent higher for persons having moderate deafness, and 35 per cent higher for those having severe deafness. Between the extremes of the first and seventh occupational classes, there is a fairly regular gradation in the influence of deafness on the unemployment rates. When consideration is given to the nature of specific occupations in each classification, it is apparent that the influence of deafness on unemployment prevalence is in direct proportion to the importance of oral communication. Without exception, in each occupational class the influence on unemployment prevalence is greater for severe deafness than for moderate deafness, and moderate deafness exerts an influence in each instance. These data support the thesis advanced in connection with Table VI above; namely, that

a. impaired hearing constitutes an unemployment risk; *b.* the degree of risk increases with stage of deafness; and *c.* the same stage of deafness varies in risk value for different occupations, depending upon the importance of good hearing in the specific occupation.

NORMAL HEARING.

So far our discussion has been concerned with characteristics and distribution of deafness cases that were reported under the classification of stage 1 deafness or worse during the household census of the National Health Survey. It was shown that these cases may be regarded as having average hearing losses of 47 dcb. or more by air conduction measurements on 1,024 and 2,048 cycles. on the basis of audiometric tests which were given to 4,721 persons who had been reported on the National Health Survey schedules as having partial deafness, stages 1, 2 or 3, or as being totally deaf for speech.

During the clinical investigation of hearing, examinations were conducted on 4,364 persons, ranging in age from 8 to 76 years, who had not been reported on the Health Survey schedules as having any stage of deafness. These persons will be referred to as the "nonreported" sample. Reasons were given above as to why it is not expected that this nonreported sample should be composed entirely, or even predominantly, of persons with normal hearing. As a matter of fact, it was found that only 38 per cent of the group (1,663 persons) had normal hearing in both ears. Audiograms were included in this normal group only if the hearing loss by air conduction was 20 dcb. or less for all eight tones on both ears. Representative ranges of auditory acuity (10 to 90 centile range) as measured by air and bone conduction on this normal group are shown in Fig. 4. The shaded areas on the graph indicate the ranges of acuity measurements that include the central 80 per cent of the observations in each distribution.

The dispersion of measurements for normal hearing by air conduction is nearly equal to that for bone conduction. The scale range delimited by the median ± 10 dcb. for distributions of measurements on this normal group is regarded in this study as defining the normal hearing characteristic for

both air and bone conduction on all tones included in the tests. Significant deviations below these limits for air conduction are regarded as hearing impairments. Deviations above or below these ranges by bone conduction are regarded as indications of better or worse than normal hearing, respectively, or as measures of prolonged or shortened bone conduction time for purposes of differential diagnosis. The audiometric zero for normal hearing by bone conduction (see Figs. 2, 5, 6 and 7) is obtained by subtracting the hearing loss level indicated by the median of this normal group (see Fig. 4, heavy black line) from the particular values under consideration.

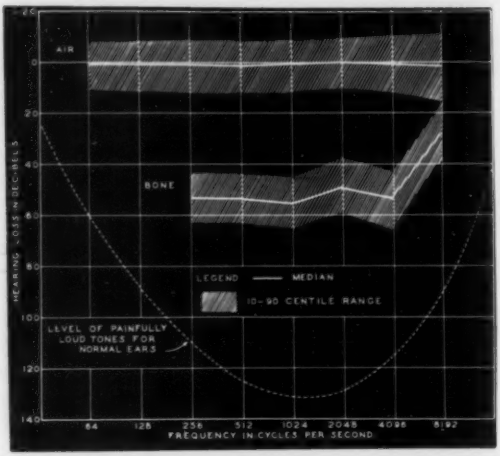

Fig. 4. Audiogram chart showing representative ranges of auditory acuity by air and bone conduction for 1,663 persons with good hearing for all tones, by air conduction in both ears.

Further discussion on the characteristics of normal hearing, based on data obtained during this study, was given by the author in a previous report (Beasley[3]). Since the actual hearing loss levels for presumably "normal hearing" groups, as determined from naive use of calibrated attenuator dials on different brands of audiometers or on different audiometers of the same brand, vary enormously in actual practice, it is important to bear in mind the nature of the normal hearing characteristic for air and bone conduction as determined from the materials in this study.

CLINICAL HISTORY OF NORMAL HEARING.

Persons giving a clinical history of normal hearing (for speech) base judgments as a rule on their personal everyday auditory experience in various listening situations, and they do not necessarily have good hearing for all tones as tested by audiometric techniqe and as defined in the preceding section. By tabulating the distributions of hearing loss measurements on persons who give a clinical history of normal hearing, we obtain evidence concerning the maximum extent to which auditory acuity may diminish before people

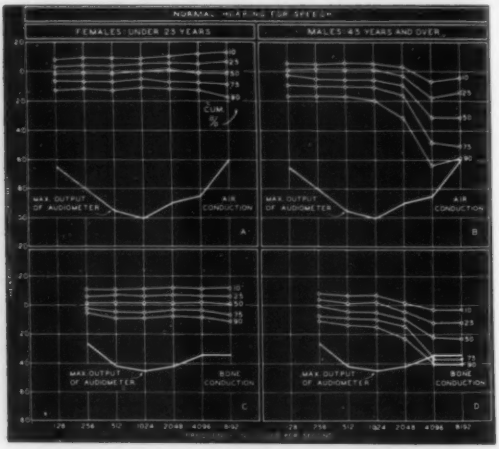

Fig. 5. Percentage audiograms showing extreme variations in auditory acuity found among persons giving a clinical history of normal hearing.

begin to notice difficulties with their hearing. In three previous reports[2, 5, 6] the writer has described in considerable detail results of this type from the present clinical investigation, showing also sex differences and variations with age. By tabulating the distributions of hearing loss measurements on persons who give a clinical history of stage 1 deafness (impairment for public address or group conversation), we gain corroborative evidence as to the levels of deafened thresholds which indicate beginnings of noticeable defects of hearing that interfere with understanding speech sounds which are from 40 to 70 dcb. above normal threshold inten-

sities. The curves in Fig. 2B above show that the average borderline for these noticeable defects of hearing is between 20 and 25 dcb. hearing loss. For this reason, a dividing line of 20 dcb. loss was chosen as the basis for defining the limits of normal hearing as described in the preceding section.

The curves in Fig. 5 show ranges of auditory acuity measurements on 1,077 females (2,154 ears) under 25 years of age and on 462 males (924 ears) 45 years of age and over who gave a clinical history of normal hearing. It is observed that more than 90 per cent of the females had normal hearing for all eight tones by air conduction and for the six tones which were employed in the bone conduction tests. Among the older males, however, there were fewer than 30 per cent who had normal hearing for all eight tones by air conduction. The region in which hearing losses are most prominent is higher in frequency than 2,048 cycles. It is clear from the character of the bone conduction losses that these deviations from good hearing norms result from nerve involvement of a circumscribed character, influencing acuity for the higher tones only (4,096 and 8,192 cycles).

One reason why severe losses on tones much lower in frequency than 4,096 cycles are not found among persons giving a clinical history of normal hearing (for speech) is that when such losses occur, say in the region of 1,024 and 2,048 cycles, the ability to perceive speech is reduced noticeably; for, as Fletcher[22] and Steinberg[32] have shown, perception of speech sounds is influenced most by elimination of frequency components in the region of 1,000 and 2,000 cycles, whereas total elimination of all components below 750 cycles or above 3,000 cycles has very little influence on the percentage error in perceiving even nonsense monosyllabic sounds. Of course, both the low and high frequency components play a rôle in the perceived *quality* of the speech sounds, but they do not determine *recognizability* to any marked extent.

There is still another reason why reduced acuity for high tones exists and yet the individual is not aware of a hearing defect. Experiments conducted, first by Fowler[27, 32] and extended later by Steinberg and Gardner,[34] show that loss of acuity for faint high tones (threshold intensities) does not indicate equivalent reduction in the loudness of the same

sounds when they are 40 to 80 dcb. above the normal threshold. The theorem has many limitations, but it apparently holds for cases of circumscribed nerve deafness affecting only the higher tones, providing the hearing is normal or nearly so for frequencies up to 2,000 cycles.

The view that reduced auditory acuity for high tones, such as that illustrated in Fig. 5, is a normal complement of advancing age has been accorded widespread acceptance; and, hence, such losses are held to be significant only when found among children or young adults. It is quite possible that just the reverse of this opinion is actually true, since the etiology and, therefore, prognosis may be entirely different for similar amounts of high tone loss at different ages. High tone "losses" arise from many different physiological, anatomical and environmental causes, and sometimes they are only artifacts of experimental technique. Certain types of losses when found in childhood *may be* symptomatic and prognostic of further progression of the impairment, whereas others are only temporary states which clear up without treatment. In many cases, minor high tone losses first appearing in the age period from 30 to 40 years are signs of a rapid progressive deafness which oftentimes runs its course in four or five years, leaving the individual definitely hard-of-hearing. In still other instances, high tone losses in middle adult years are simply results of exposure to concussional shock, to percussional noise, or to prolonged high noise level over a period of years. In none of these three cases is there expectancy for progression in the impairment. In any particular case, prognosis in respect to high tone losses depends directly not on amount of loss nor age of patient but on the whole *syndrome*, which as yet cannot be defined clearly for clinical purposes. There remains also the important question as to whether *super* high tone losses (sounds above 10,000 cycles per second) which have been found with high prevalence among school children,[17, 18, 19, 20] and which are alleged to be due to *conductive* lesions only, bear any direct relationship to high tone losses in the range from 3,500 to 8,000 cycles, which are found less often among school children and more often during middle adult years and which are demonstrably associated with *nerve* lesions. It appears that several syndromes must be formulated, and prognosis will differ for each one.

CHARACTERISTICS OF HEARING LOSS IN STAGE 2 AND STAGE 3 DEAFNESS.

The types of audiograms and levels of hearing loss found among persons having a social history (household census) of any degree of partial deafness (stages 1, 2 or 3) are represented approximately by the varieties of audiograms obtained for persons having a clinical history of stage 2 and stage 3 deafness. Individual cases, selected on the basis of being typical in the various age and sex groups, are shown in Figs. 6 and 7. These audiogram patterns fall into three broad groups: *a*. those which are approximately horizontal, peaked toward the center, or rise gradually from low to high tones; *b*. those which descend gradually from low to high tones, or show only 10 to 20 dcb. more loss for high tones than for low tones; and *c*. those which descend steeply from low to high tones, showing 25 to 35 dcb. or more excess loss on high tones, involving relatively greater losses on 1,024 and 2,048 cycles than on tones lower in pitch (512 cycles or less). This grouping uses as a point of reference the relative prominence of hearing losses for tones higher in pitch than 2,048 cycles, and whether or not the hearing losses are relatively greater for frequencies most important for speech perception (1,024 and 2,048 cycles) than for the lower tones (64 to 512 cycles).

DISTRIBUTION OF MINOR AND ADVANCED STAGES OF HIGH TONE LOSS IN THE GENERAL POPULATION.

During the clinical investigation of hearing, examinations were conducted on 4,364 persons of all ages who had not been reported on the Health Survey as having any stage of deafness. By the random fashion in which these persons were obtained as subjects for the clinical study, it is considered that they are representative of the nonreported sample. Hearing loss measurements obtained for these persons, when weighted in accordance with the prevalence rate of normal hearing on the Health Survey enumeration, and when added onto the hearing losses obtained for persons in the various classes of partial and total deafness for speech, provide a basis for constructing a hearing loss scale which applies to a general trend in the population at each age level.

In order to derive an estimate as to the prevalence of high tone deafness in the Health Survey normal group, the following simple classification of audiogram types was adopted: All of the audiograms in this "normal" group (4,364 persons) were classified according to the average hearing loss on 256, 512 and 1,024 cycles. Group 1, an average of 15 dcb.

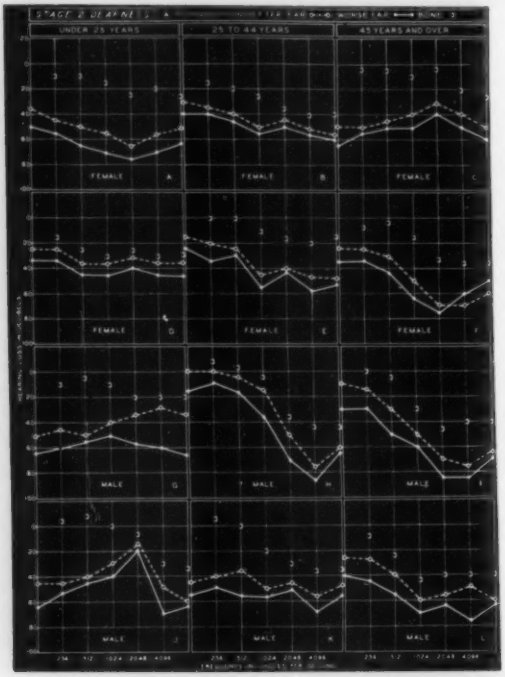

Fig. 6. Individual cases illustrating the various patterns of hearing loss found among persons giving a clinical history of stage 2 deafness.

or less on these three tones; group 2, 15.1 to 30 dcb. average loss on these three tones; and group 3, 30.1 dcb. or more average loss on these three tones. Each of these three groups was then subdivided in terms of characteristics of the audiogram on 2,048, 4,096 and 8,192 cycles. These subgroups are designated as A, B and C. Subgroup A designates audiograms that were approximately level, peaked or ascending

on these three high tones in relation to the average loss on 256, 512 and 1,024 cycles. Subgroup B designates audiograms that show moderate high tone loss, with hearing losses on 2,048 cycles and above, descending on the average of 10 to 25 dcb. below the average loss on 256, 512 and 1,024 cycles. Subgroup C designates advanced high tone loss where the

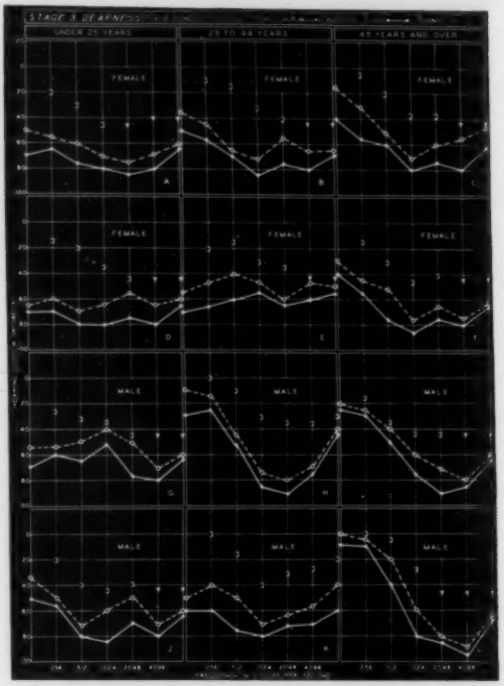

Fig. 7. Individual cases illustrating the various patterns of hearing loss found among persons giving a clinical history of stage 3 deafness.

audiogram slopes steeply downward from low to high tones, with the hearing losses for 1,024 and 2,048 cycles being 15 dcb. or more in excess of the average on 256, 512 and 1,024 cycles. The percentage distribution of these nine groups of audiograms was calculated at each decade for males and females in the Health Survey normal sample. These percentage distributions were then weighted in terms of the

prevalence rate for Health Survey normal hearing. These weighted percentage distributions, when added onto prevalence rates for all stages of deafness reported on the Health Survey, yield, then, a total of 100 per cent at each age level. The distribution of these audiogram types, in relation to age and sex and in relation to persons having stage 1 deafness or worse, as reported on the Health Survey, are shown in Table VIII.

Two assumptions, which are implicit both in the construction of the distributions of prevalence rates (see Table VIII) and in the analysis of trends shown by the table, are the following: *a.* that the sample of 4,364 persons who were studied at the clinic and who had not been reported on the Health Survey as having any impairment of hearing is *randomly* representative of the rest of the people — those who were not studied at the clinic and who, likewise, were not reported on the Health Survey as having any impairment of hearing; *b.* that each age group as classified in Table VIII is *randomly* representative of similar age groups in the general population in respect to the prevalence in kind and amount of hearing loss among them. One cannot *prove* the validity of either assumption. Degree of error in the resulting distributions presented in Table VIII can be *estimated* through repeated surveys of the type described in this report, providing due consideration is given always to comparability of technique. After careful study of the conditions under which these samples of the population were obtained, the writer has been unable to discover any factor that would bias the proportions shown in Table VIII to any marked extent. For purposes of discussion, then, the above two assumptions will be reviewed as tenable.

The figures in Table VIII show the estimated prevalence (in per cent) for each type of hearing condition defined above at successive 10-year age periods, beginning with the group 5 to 14 years, and for the two sexes separately. Interpretation of the trends shown by the table is facilitated by inspection of the curves in Fig. 8.

The major interest underlying the classification of audiogram types employed in this analysis is to reveal the relative prominence of minor and advanced stages of high tone loss at various decades of life among both males and females, and

TABLE VIII.

Estimated Prevalence Rates for Various Levels of Hearing Loss and Relative Degrees of High Tone Loss, According to Age and Sex. Estimates Apply to General Population (See Text Matter).

Classification* of Hearing Condition	Percentage Distribution for Various Hearing Conditions — Age Range in Years						
	5-14	15-24	25-34	35-44	45-54	55-64	65 & Over
Male							
All groups	100.00	100.00	100.00	100.00	100.00	100.00	100.00
Not reported on Health Survey	99.70	99.65	99.52	99.03	98.51	97.07	89.73
Group 1, all	85.39	86.88	82.53	74.16	66.80	46.80	21.79
1A	51.81	46.14	32.06	17.06	5.84	1.10	0.44
1B	18.86	23.25	23.63	21.23	14.61	4.24	1.33
1C	14.72	17.49	26.84	35.87	46.35	41.46	20.02
Group 2, all	11.08	8.14	10.47	16.37	19.20	30.94	34.73
2A	7.86	5.25	5.60	5.56	4.28	1.88	1.19
2B	1.98	0.83	2.18	4.09	4.27	5.34	1.78
2C	1.24	2.06	2.69	6.72	10.65	23.72	31.76
Group 3, all	3.23	4.63	6.52	8.50	12.51	19.33	33.21
3A	1.40	2.16	3.00	2.01	1.77	1.10	0.15
3B	0.83	1.54	0.93	2.32	1.77	2.83	2.22
3C	1.00	0.93	2.59	4.17	8.97	15.40	30.84
Reported on Health Survey, Stage 1 or worse	0.30	0.35	0.48	0.97	1.49	2.93	10.27
Female							
All groups	100.00	100.00	100.00	100.00	100.00	100.00	100.00
Not reported on Health Survey	99.77	99.71	99.50	99.07	98.45	97.36	92.07
Group 1, all	84.30	85.28	79.23	71.12	57.53	43.06	20.13
1A	61.47	57.22	47.87	35.97	18.23	6.03	0.24
1B	12.65	16.13	18.01	20.19	19.16	14.65	5.21
1C	10.18	11.93	13.35	14.96	20.14	22.38	14.68
Group 2, all	11.54	9.35	12.22	16.65	28.18	35.17	35.98
2A	9.83	8.06	9.57	12.04	16.09	10.86	6.15
2B	1.11	0.40	1.58	2.59	5.95	10.69	10.42
2C	0.60	0.89	1.07	2.02	6.14	13.62	19.41
Group 3, all	3.93	5.08	8.05	11.30	12.74	19.13	35.96
3A	2.56	2.90	4.72	4.44	4.37	3.79	3.79
3B	0.60	0.98	1.57	3.15	3.72	4.48	7.34
3C	0.77	1.20	1.76	3.71	4.65	10.86	24.83
Reported on Health Survey, Stage 1 or worse	0.23	0.29	0.50	0.93	1.55	2.64	7.93

*See text matter for explanation of classifications.

to determine whether there is a demonstrable relationship between minor high tone losses at a given age and more advanced stages at a later age. Reading across any given row of Table VIII, one observes how the prevalence of that hearing condition *changes* in successively older groups of people. Reading down each column of figures, one is able to determine what type (or types) of hearing predominates during any given age period, among either males or females. By comparing *amounts* and *sign* (decrement or increment) of differences between prevalence rates for each type of hearing

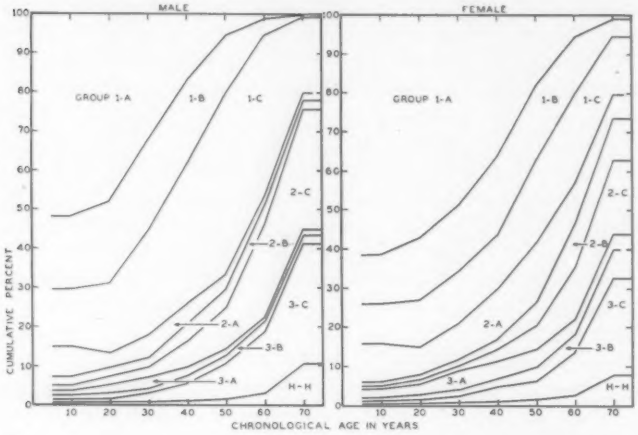

Fig. 8. Cumulative per cent curves showing prevalence at successive decades of various conditions of hearing (defined in text matter). Group H-H are those reported on National Health Survey as stage 1 deafness or worse.

in successive pairs of age groups, one can deduce which groups in one age period contribute most to marked changes in prevalence in the next older group.

The "A" groups are composed of audiograms which are approximately level, peaked in center, or rise gradually from low to high tones. Group 1A contains only audiograms which represent good hearing for all eight tones (15 dcb. or better). It is observed that during the age period 5 to 14 years, group 1A audiograms represent the hearing of 51.8 per cent of the males and 61.5 per cent of the females. The prevalence of this type of hearing ability diminishes most rapidly through

the age period 25 to 44 years. In the period 45 to 54 years, 5.8 per cent of males and 18.2 per cent of females have good hearing for all tones.

There is not a conspicuous change in the prevalence of either 2A and 3A types of audiograms in successive decades. Among males, the prevalence of 2A audiograms changes continuously from 7.9 per cent at ages 5 to 14 years to 1.2 per cent at 65 years and over; whereas the prevalence of 3A audiograms changes from 1.4 per cent at 5 to 14 years to 3 per cent at 25 to 34 years, thereafter declining to 0.2 per cent at 65 years and over. Among females, the prevalence of 2A audiograms remains quite constantly at about 9 per cent through the age range 5 to 34 years, rises to 16.1 per cent at 45 to 54 years, and falls off to 6.2 per cent at 65 years and over; whereas the prevalence of 3A audiograms rises from 2.6 per cent at 5 to 14 years to 4.7 per cent at 25 to 34 years, thereafter declining gradually to 3.9 per cent at 65 years and over.

The course of changes with advancing age in the prevalence of type B audiograms (minor or gradual high tone loss) behaves quite differently among males and females. The most conspicuous variation in the prevalence of this type of hearing is the rapid decline in the prevalence of 1B among males from 23.6 per cent at 25 to 34 years to 1.3 per cent at 65 years and over. Type 2B rises gradually from 2 per cent at 5 to 14 years to 5.3 per cent at 55 to 64 years. Type 3B varies irregularly and insignificantly from a minimum of 0.8 per cent to 2.8 per cent at various ages. Among females, 1B audiograms increase in prevalence from 12.7 per cent at 5 to 14 years to 20.2 per cent at 35 to 44 years, thereafter declining to 5.2 per cent at 65 years and over. Type 2B increases gradually in prevalence from 1.1 per cent at 5 to 14 years to 10.4 per cent at 65 years and over. Type 3B increases gradually from 0.6 per cent at 5 to 14 years to 7.3 per cent at 65 years and over.

By far the most conspicuous changes at successive ages among both males and females occur in the prevalence of 1A audiograms (rapid and continuous decrement). 1C audiograms (rapid increment followed by marked decrement), 2C and 3C audiograms (continuous and marked rate of increment at all ages). The magnitudes and sign of the changes

in prevalence of these several types of audiograms are of such a character that a *necessary* conclusion results: *certain levels and degrees of high tone loss in one decade are symptomatic precursors of greater degrees of the same type of loss during subsequent decades.*

In order to facilitate observation of the changes from decade to decade, which lead to this important conclusion, differences in the prevalence rates for each type of hearing condition as given in Table VIII are assembled in Table IX.

The first column of figures in Table IX shows the differences between prevalence rates for each type of hearing condition in the age groups 5 to 14 years and 15 to 24 years. It is assumed that the prevalence rates (see Table VIII) may be regarded as representing conditions of hearing for persons at the median age of the group (*i.e.*, at 10 years, for the 5- to 14-year-old group; at 20 years, for the 15- to 24-year-old group; and so on, for the other age groups). It is assumed, further, that the change represented by the differences between the prevalence rates occurred during the age period designated by two successive median ages (*i.e.*, from 10 to 20 years for the two age groups 5 to 14 years and 15 to 24 years). The sign of the difference between two prevalence rates is positive when the rate is higher for the older group. Table IX should be read in conjunction with Table VIII, since interpretation of the significance of various amounts of decrement or increment in successive age groups depends not only on the amount and sign of the difference but also on the values of the prevalence rates involved. The values given in Table IX, when divided by 10, can be read as average annual incidence of new cases per 100 population at each single year throughout the decade period indicated.

The comparisons which can be made between the differences in Table VI and the rates in Table V are too numerous to review in detail; consequently, the events in only one age period will be examined for purposes of illustrating the method of analysis.

Consider, first, the period 30 to 39 years for males, which shows differences between prevalence rates for the various hearing conditions in the age groups 25 to 34 years and 35 to 44 years. Group 1 audiograms (all subclasses) decline in

prevalence rate to the extent of 8.37. This decline is offset by increments of 5.90 in group 2, 1.98 in group 3, and 0.49

TABLE IX.

Differences Between Per Cent Prevalence Rates (Table V) for Various Conditions of Hearing in Successive 10-Year Groups.

Classification* of Hearing Condition	Difference Between Per Cent Prevalence Rates for Successive Decades					
	Assumed Age Period in Years					
	10-19	20-29	30-39	40-49	50-59	60 & Over
	Male					
Not reported on Health Survey	—0.05	—0.13	—0.49	—0.52	—1.44	—7.34
Group 1, all	+1.49	—4.35	—8.37	—7.36	—20.00	—25.01
Group 2, all	—2.94	+2.33	+5.90	+2.83	+11.74	+3.79
Group 3, all	+1.40	+1.89	+1.98	+4.01	+6.82	+13.88
Group 1A	—5.67	—14.08	—15.00	—11.22	—4.74	—0.66
Group 2A	—2.61	+0.35	—0.04	—1.28	—2.40	—0.69
Group 3A	+0.76	+0.84	—0.99	—0.24	—0.67	—0.95
Group 1B	+4.39	+0.38	—2.40	—6.62	—10.37	—2.91
Group 2B	—1.15	+1.35	+1.91	+0.18	+1.07	—3.56
Group 3B	+0.71	—0.61	+1.39	—0.55	+1.06	—0.61
Group 1C	+2.77	+9.35	+9.03	+10.48	—4.89	—21.44
Group 2C	+0.82	+0.63	+4.03	+3.93	+13.07	+8.04
Group 3C	—0.07	+1.66	+1.58	+4.80	+6.43	+15.44
Reported on Health Survey, Stage 1 or worse	+0.05	+0.13	+0.49	+0.52	+1.44	+7.34
	Female					
Not reported on Health Survey	—0.06	—0.21	—0.43	—0.62	—1.09	—5.29
Group 1, all	+0.98	—6.05	—8.11	—13.59	—14.47	—22.93
Group 2, all	—2.19	+2.87	+4.43	+11.53	+6.99	+0.81
Group 3, all	+1.05	+2.97	+3.25	+1.44	+6.39	+16.83
Group 1A	—4.25	—9.35	—11.90	—17.74	—12.20	—5.79
Group 2A	—1.77	+1.51	+2.47	+3.95	—5.23	—4.71
Group 3A	+0.34	+1.82	—0.28	—0.07	+0.58	0.00
Group 1B	+3.48	+1.88	+2.18	—1.03	—4.51	—9.44
Group 2B	—0.71	+1.18	+1.01	+3.36	+4.74	—0.27
Group 3B	+0.38	+0.59	+1.58	+0.57	+0.76	+2.86
Group 1C	+1.75	+1.42	+1.61	+5.18	+2.24	—7.70
Group 2C	+0.29	+0.18	+0.95	+4.12	+7.48	+5.79
Group 3C	+0.43	+0.56	+1.95	+0.94	+6.21	+13.97
Reported on Health Survey, Stage 1 or worse	+0.06	+0.21	+0.43	+0.62	+1.09	+5.29

*See text matter for explanation of classifications.

in stage 1 or worse (total of these increments is 8.37). In any given age distribution the decrements and increments balance. It is not a necessary conclusion, however, that the decrement of 8.37 in group 1 is absorbed in groups 2, 3 and stage 1 or worse in the proportion of the increments indicated. The same balance would be obtained if all cases represented by the decrement 8.37 in group 1 became group 2 cases during this period, and a sufficient number of group 2 cases became group 3 or stage 1 or worse. Greater certainty of conclusions regarding specific group changes can be achieved by taking account of differences in the subclasses of each group.

Consider changes in the "A" groups for males. Group 1A shows a decrement of 15.0, from a prevalence of 32.1 at 30 years to 17.1 at 40 years (age medians). What becomes of these cases? They must fall into groups 1B or 1C, group 2, group 3, or stage 1 or worse. They do not become 1B, 2A or 3A, since all three of these groups show a slight decrement during this period rather than increment, and the total quantity of cases involved in these declining groups is too small to balance the decrements from group 1A. The 1A decrement must be absorbed largely into 1C and 2C, since these are the only groups which show any appreciable increment during this period. The combined increment in all other groups is offset by the decrements in groups 1B, 2A and 3A. It is apparent, therefore, that the majority of cases which drop out of group 1A during the period 30 to 39 years become cases of advanced high tone loss as designated by criteria for groups 1C and 2C.

A perusal of the signs and magnitudes of change among males in the subclasses for other age periods leads to some interesting deductions. After the period 20 to 29 years, all of the "A" groups show continuous decrement, the highest rate of change being in group 1A. Similarly, after the period 20 to 29 years, the 1B group shows continuous decrement, the most active periods of change being from 40 to 59 years. This means that males having good hearing for all tones, or only minor high tone losses, progress to either 2B, 3B, or 1C, 2C or 3C groups after 30 years of age, or become hard-of-hearing (stage 1 or worse). An examination of the changes in the 2B and 3B groups for these later age periods indicates

that the increments are far too small to balance the decrements in the better hearing groups. The large gains are in the "C" groups, which represent advanced high tone loss involving frequencies in the speech range (1,024 and 2,048 cycles).

The sequence of increments and decrements in the 1C, 2C and 3C groups is likewise indicative of progressive nerve deafness. Group 1C gains in prevalence throughout the period 10 to 49 years, after which it declines. Group 2C gains in prevalence, at a slower rate than 1C, throughout this same period, then shows a sharp rise in prevalence during the subsequent periods when 1C is declining. Group 3C lags the increasing prevalence of 2C cases and shows a sharp increase after 60 years, when the gain in 2C cases is less than during the preceding decade, even though 1C declines considerably more after 60 years than 2C gains.

These sequences justify only one final conclusion: that loss of good hearing with advancing age is balanced chiefly by increases in the prevalence of chronic progressive nerve deafness, and that moderate degrees of high tone loss in one decade are symptomatic indications of more severe and continuously progressive high tone loss for the subsequent decade. The trends reviewed apply directly to males, but a careful examination of the portion of the tables which present similar data for females will reveal equivalent processes. The major difference between the sexes is that "B" groups (gradual or minor high tone losses) assume relatively greater prominence among females, as transition stages, and "C" groups do not attain as high a prevalence at any age among females as among males.

REFERENCES.

1. Beasley, Willis C.: Significance, Scope and Method of a Clinical Investigation of Hearing in the General Population. U. S. Public Health Service, Washington, 1938 (Bulletin No. 1, Hearing Study Series, National Health Survey Preliminary Reports).

2. Beasley, Willis C.: Preliminary Analysis of Audiometric Data in Relation to Clinical History of Impaired Hearing. U. S. Public Health Service, Washington, 1938 (Bulletin No. 2, Hearing Study Series, National Health Survey Preliminary Reports).

3. Beasley, Willis C.: Prevalence of Aural Pathology and Clinical History of Impaired Hearing Among Males and Females of Various Ages. U. S. Public Health Service, Washington, 1938 (Bulletin No. 3, Hearing Study Series, National Health Survey Preliminary Reports).

4. BEASLEY, WILLIS C.: Normal Hearing by Air and Bone Conduction. U. S. Public Health Service, Washington, 1938 (Bulletin No. 4, Hearing Study Series, National Health Survey Preliminary Reports).

5. BEASLEY, WILLIS C.: Normal Hearing for Speech at Each Decade of Life. U. S. Public Health Service, Washington, 1938 (Bulletin No. 5, Hearing Study Series, National Health Survey Preliminary Reports).

6. BEASLEY, WILLIS C.: Sex Differences and Age Variations in Hearing Loss in Relation to Stage of Deafness. U. S. Public Health Service, Washington, 1938 (Bulletin No. 6, Hearing Study Series, National Health Survey Preliminary Reports).

7. BEASLEY, WILLIS C.: Generalized Age and Sex Trends in Hearing Loss. U. S. Public Health Service, Washington, 1938 (Bulletin No. 7, Hearing Study Series, National Health Survey Preliminary Reports).

8. BEASLEY, WILLIS C.: Partial Deafness and Hearing-Aid Design: I. Characteristics of Hearing Loss in Various Types of Deafness. *Jour. Soc. Mot. Pict. Eng.*, 35:59-85, 1940.

9. BEASLEY, WILLIS C.: Correlation Between Hearing Loss Measurements by Air Conduction on Eight Tones. *Jour. Acous. Soc. Amer.*, 12:104-113, 1940.

10. BEASLEY, WILLIS C.: Characteristics and Distribution of Impaired Hearing in the Population of the United States. *Jour. Acous. Soc. Amer.*, 12:114-121, 1940.

11. BORDLEY, JOHN E., and HARDY, MARY: Effect of Lesions of the Tympanic Membrane on the Hearing Acuity. *Arch. Otolaryngol.*, 26:649-657, 1937.

12. BRITTEN, R. H.; COLLINS, S. D., and FITZGERALD, J. S.: The National Health Survey: Some General Findings as to Disease, Accidents and Impairments in Urban Areas. *Pub. Health Rep.*, 55:444-470, 1940.

13. BUNCH, C. C.: The Diagnosis of Occupational or Traumatic Deafness; a Historical and Audiometric Study. THE LARYNGOSCOPE, 47:615-691, 1937.

14. CIOCCO, A.: Observations on the Hearing of 1980 Individuals: a Biometric Study. THE LARYNGOSCOPE, Nov., 1932.

15. CIOCCO, A.: The Consistency and Significance of Tests Made with a 4A Audiometer. *Pub. Health Rep.*, 51:1402-1416, 1936.

16. CIOCCO, A.: Variations in Auditory Acuity of 543 School Children Re-examined After an Average Interval of Three Years. *Ann. Otol., Rhinol. and Laryngol.*, 46:55-69, 1937.

17. CROWE, S. J.: The Prevention of Deafness in Children. THE LARYNGOSCOPE, July, 1939.

18. CROWE, S. J.; HUGHSON, W., and WITTING, E. G.: Function of the Tensor Tympani Muscle. *Arch. Otolaryngol.*, 14:575-580, 1931.

19. CROWE, S. J., and GUILD, S. R.: Impaired Hearing for High Tones. *Acta Oto-Laryngol.*, 26:138-144, 1938.

20. CROWE, S. J., and BAYLOR, J. W.: The Prevention of Deafness. *Jour. A. M. A.*, 112:585-590, 1939.

21. FLETCHER, H.: New Methods and Apparatus for Testing Hearing. *Ann. Otol.*, 35:165-180, 1926.

22. FLETCHER, H.: Speech and Hearing. Van Nostrand, New York, 1929.

23. FLETCHER, H.: Physical Characteristics of Speech and Music. *Bell Sys. Tech. Jour.*, 10:349-373, 1931.

24. FLETCHER, H., and MUNSON, W. A.: Loudness: Its Definition, Measurement and Calculation. *Jour. Acous. Soc. Amer.*, 5:82-108, 1933.

25. FOWLER, E. P.: Deafness in School Children. *Arch. Otolaryngol.*, 6:43-53, 1927.

26. FOWLER, E. P.: Theoretical Interpretations of the Functional Tests. *Arch. Otolaryngol.*, 7:606-613, 1928.

27. FOWLER, E. P.: Marked Deafened Areas in Normal Ears. *Arch. Otolaryngol.*, 8:151-155, 1928.

28. FOWLER, E. P.: Otosclerosis Complicated by Other Lesions: a Study of Roentgenograms, Audiograms, Laboratory and Clinical Findings. *Ann. Otol., Rhinol. and Laryngol.*, 42:714-741, 1933.

29. FOWLER, E. P.: The Effect of Severe Illness Upon the Hearing. *Ann. Otol., Rhinol. and Laryngol.*, 43:387-401, 1934.

30. FOWLER, E. P.: Ear Conditions from the Preventive Standpoint. *Health Exam.*, Oct., 1935.

31. FOWLER, E. P.: A Method for the Early Detection of Otosclerosis. *Arch. Otolaryngol.*, 24:731-741, 1936.

32. FOWLER, E. P.: The Diagnosis of Diseases of the Neural Mechanism of Hearing by the Aid of Sounds Well Above Threshold. THE LARYNGOSCOPE, 47:289-300, 1937.

33. FOWLER, E. P., and FLETCHER, H.: Three Million Deafened School Children. *Jour. A. M. A.*, 87:1877-1882, 1926.

34. FROST, W. H.: Epidemiology. Nelson Loose-Leaf Living Med., 7:3,91-109.

35. GEIGER, J. C.; RODIN, F. H., and CHASE, B. A.: The Problem of the Hard-of-Hearing School Child. *Arch. Pediat.* 56:605-610, 1939.

36. GREENWOOD, MAJOR: Epidemiology: Historical and Experimental. London, Oxford University Press, 1932.

37. GREENWOOD, MAJOR: Epidemics and Crowd Diseases. New York, Macmillan, 1935.

38. GUILD, S. R.: A Method of Classifying Audiograms. THE LARYNGOSCOPE, Nov., 1932.

39. GUILD, S. R.: Symposium on Tone Localization in the Cochlea. (American Otological Society, May, 1935.) *Ann. Otol., Rhinol. and Laryngol.*, 44:739-754, 1935.

40. GUILD, S. R.: Symposium on Hearing by Bone Conduction. (American Otological Society, May, 1936.) *Ann. Otol., Rhinol. and Laryngol.*, 45:737-755, 1936.

41. GUILD, S. R.; CROWE, S. J.; BUNCH, C. C., and POLVOGT, L. M.: Correlations of Differences in the Density of Innervation of the Organ of Corti with Differences in the Acuity of Hearing, Including Evidence as to the Location in the Human Cochlea of the Receptors for Certain Tones. *Acta Oto-Laryngol.*, 15:269-308, 1931.

42. GUILDER, RUTH P., and HOPKINS, LOUISE A.: Auditory Function Studies in an Unselected Group of Pupils at the Clarke School for the Deaf. THE LARYNGOSCOPE, Jan., Feb. and March, 1936.

43. HUGHSON, W.: Grafts in the Round Windows in the Treatment of Certain Types of Deafness. *Arch. Otolaryngol.*, 25:623-631, 1937.

44. HUGHSON, W., and CROWE, S. J.: Function of the Round Window. *Jour. A. M. A.*, 96:2027-2028, 1931.

45. HUGHSON, W.; THOMPSON, E., and WITTING, E. G.: An Experimental Study of Bone Conduction. *Ann. Otol., Rhinol. and Laryngol.*, 45:844-859, 1936.

46. KNUDSEN, VERN O.: Interfering Effect of Tones and Noise Upon Speech Perception. *Phys. Rev.*, 26:133-138, 1926.

47. KNUDSEN, VERN O., and JONES, I. H.: A Plan for Group Testing of School Children. *Ann. Otol.*, 35:817-821, 1926.

48. LILLIE, H. I., and KRANZ, F. W.: A Quantitative Study of Hearing With and Without Cotton Plug Protheses in the Middle Ear. *Ann. Otol.*, 33:458-472, 1924.

49. MARTENS, ELISE H.: The Deaf and Hard-of-Hearing in the Occupational World. Bulletin, 1936, No. 13, U. S. Dept. of the Interior, Washington, 1937.

50. NEWHART, HORACE: Diagnostic School Clinic in the Public Schools as a Factor in Conservation of Hearing. *Jour. A. M. A.*, 87:1882-1885, 1926.

51. NEWHART, HORACE: The Results of a Preliminary Survey of School Children by Audiometric Methods. *Trans. Amer. Laryngol., Rhinol. and Otol. Soc.*, 1926.

52. NEWHART, HORACE: Progress in the Prevention of Deafness. *Jour. A. M. A.*, 93:1117-1119, 1929.

53. NEWHART, HORACE: The Early Detection and Prevention of Hearing Impairment. *Trans. Amer. Child Health Assn.*, 1929.

54. ODA, D.: Observations on the Pathology of Impaired Hearing for Low Tones. THE LARYNGOSCOPE, Nov., 1938.

55. PERRETT, G. ST. J.; TIBBITTS, C., and BRITTEN, R. H.: The National Health Survey: Scope and Method of the Nationwide Canvass of Sickness in Relation to Its Social and Economic Setting. *Pub. Health Rep.*, 54:1663-1687, 1939.

56. POHLMAN, A. G.: The Problem of Middle Ear Mechanics. *Ann. Otol., Rhinol. and Laryngol.*, June, 1922.

57. POHLMAN, A. G.: Theories of Sound Analysis and Intensity Control on the Basis of Middle and Inner Ear Mechanics. *Ann. Otol., Rhinol. and Laryngol.*, Sept., 1927.

58. POHLMAN, A. G.: The Interpretation of Conduction Deafness. *Arch. Otolaryngol.*, 14:48-63, 1931.

59. POHLMAN, A. G.: Possibilities in Quantitative Correlation Between Air and Bone Transmitted Speech. THE LARYNGOSCOPE, March, 1931.

60. POHLMAN, A. G.: A Reconsideration of the Mechanics of the Auditory Apparatus. *Jour. Laryngol. and Otol.*, 48:156-195, 1933.

61. POHLMAN, A. G.: The Mechanics of the Middle Ear Prosthesis. *Ann. Otol., Rhinol. and Laryngol.*, June, 1936.

62. POHLMAN, A. G.: The Present Status of the Mechanics of Sound Conduction in Its Relation to the Possible Correction of Conduction Deafness. *Jour. Acous. Soc. Amer.*, 8:112-117, 1936.

63. POHLMAN, A. G., and KRANZ, F. W.: Quantitative Tests on Bone and Air Acuity in Paracusis Willisi. *Arch. Otolaryngol.*, 3:136-143, 1926.

64. POHLMAN, A. G., and KRANZ, F. W.: Is a Differential Diagnosis Between Middle and Internal Ear Deafness Possible? THE LARYNGOSCOPE, April, 1926.

65. POHLMAN, A. G., and KRANZ, F. W.: Monaural Tests on Bone Acuity Under Various Conditions, with Some General Comments on Bone Acuity. *Ann. Otol., Rhinol. and Laryngol.*, Sept., 1926.

66. POHLMAN, A. G., and KRANZ, F. W.: Observations on Hearing Acuity for Bone Transmitted Sound. *Proc. Soc. for Exp. Biol. and Med.*, 24:456-461, 1927.

67. POLVOGT, L. M., and BORDLEY, J. E.: Pathologic Changes in the Middle Ear of Patients with Normal Hearing and of Patients with a Conduction Type of Deafness. *Ann. Otol., Rhinol. and Laryngol.*, 45:760-769, 1936.

68. SCAL, J. COLEMAN: Noise Deafness in Industry and Environment. *N. Y. State Jour. Med.*, Nov. 1, 1933.

69. SHAMBAUGH, G. E.: Explanation for the Symptom of Paracusis Willisi. *Arch. Otolaryngol.*, 6:228-236, 1927.

70. SHAMBAUGH, G. E.: Diseases of the Ear in Childhood. *Amer. Jour. Dis. Child.*, 39:1062-1067, 1930.

71. SHAMBAUGH, G. E.: The Problems of Deafness. *Arch. Otolaryngol.*, 14:36-47, 1931.

72. SHAMBAUGH, G. E.; HALL, A. K.; HAGENS, E. W., and HOLDERMAN, J. W.: Supplementary Statistical Studies of the Children in the Chicago Public Schools for the Deaf. *Arch. Otolaryngol.*, 5:209-219, 1927.

73. SHAMBAUGH, G. E.; HAGENS, E. W.; HOLDERMAN, J. W., and WATKINS, R. W.: Physical Causes of Deafness. *Arch. Otolaryngol.*, 7:424-513, 1928.

74. SHAMBAUGH, G. E.; HAYDEN, D. B.; HAGENS, E. W., and WATKINS, R. W.: Statistical Studies of Children in Public Schools for the Deaf. *Arch. Otolaryngol.*, 12:190-245, 1930.

75. SHAMBAUGH, G. E.; WALLNER, L. J.; GREENE, L. D., and SHAMBAUGH, G. E., JR.: Severe Deafness in Adults . *Arch. Otolaryngol.*, 18:430-448, 1933.

76. SHAMBAUGH, G. E., JR.: The Prevention and Treatment of Deafness. *Ann. Otol., Rhinol. and Laryngol.*, 43:513-525, 1934.

77. SHAMBAUGH, G. E., JR.: Interpretation of Bone Conduction and Other Tests of Hearing. *Ann. Otol., Rhinol. and Laryngol.*, 45:780-798, 1936.

78. SMYTH, H. F.: Noise in Industry: Its Effect on the Hearing and on General Health. *Ann. Otol.*, 41:1108-1115, 1932.

79. SONNENSCHEIN, R.: Some Points in the Observation of Children Who Are Deaf or Hard-of-Hearing. ORALISM AND AURALISM, July, 1924.

80. SONNENSCHEIN, R.: Functional Examination of Hearing; Deafmutism and Education of the Deaf. *Arch. Otolaryngol.*, 13:98-132, 1931.

81. SONNENSCHEIN, R., and LESHIN, N.: Functional Tests of Hearing. *Arch. Otolaryngol.*, 19:70-98, 1934.

82. STEINBERG, J. C.: Effects of Distortion on the Recognition of Speech Sounds. *Jour. Acous. Soc. Amer.*, 1:121-137, 1929.

83. STEINBERG, J. C.: Symposium on Tone Localization in the Cochlea. (American Otological Society, May, 1935.) *Ann. Otol., Rhinol. and Laryngol.*, 44:820-823, 1935.

84. STEINBERG, J. C., and GARDNER, M. B.: The Dependence of Hearing Impairment on Sound Intensity. *Jour. Acous. Soc. Amer.*, 9:11-23, 1937.

85. THEOBALD, J. J.: Conditions of Hearing Found in 27,978 Chicago Public School Children. *Arch. Otolaryngol.*, 9:686-688, 1929.

OTITIC HYDROCEPHALUS. REPORT OF TWO CASES.*

DR. J. J. GITT, St. Louis.

Otitic hydrocephalus is one of the rare complications of nasal sinus infections. This report of two cases is presented to aid in the clinical recognition of this syndrome.

Symonds[1] focused attention on this condition and gave the clinical and laboratory differentation as follows: *1.* Age; almost always children and adolescents. *2.* General condition; between the attacks of headache the patient feels and looks well. *3.* Fundi; papilloedema constant; usually measurable swelling with hemorrhages and exudate. *4.* Localizing signs; no localizing signs are present. A sixth nerve paralysis on the side of the lesions is present in a fair number of cases. Rarely unilateral or bilateral convulsive seizures may occur, or the plantar response on one or both sides may be extensor — the result, presumably, of increased intracranial pressure. *5.* C.S.F.; clear; pressure often above 300 mm.; quantity abundant; no excess of protein or cells. He pointed out that this was no new nosological entity, having been commented on by Taylor in 1890, and suggested "Otitic Hydrocephalus" to designate this syndrome.

The physiopathology is not definitely known. Kelly[2] quotes the theories held as follows: that Quincke suggested there was a simple serous inflammation of the ventricles with an increase in secretion of C.S.F. by the choroid plexus; that Bateman held there was a deficient absorption by the arachnoid villi; that Symonds thought there was an obstructive internal hydrocephalus with occlusion of the outlets for the C.S.F.; that Fanconi compromised, in that in some cases there was a hyposecretion and in others a hypersecretion of C.S.F.

Since Symonds' report, there have appeared cases by Symonds,[2] Scal,[3] Garland and Seed,[4] Smith,[5] Goodhart and Savitsky,[6] Ersner and Myers,[7] Bradheer and Gibson,[8] Wil-

*From the Department of Neurology (Washington University Medical Unit), St. Louis City Hospital, No. 1, and Jewish Hospital, St. Louis, Mo.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Jan. 25, 1940.

liams[9] (an excellent review), Kessler and Savitsky[10] and Kelly.[11]

Case 1: K. Z. (Reg. No. 103538), age 23 years, white, female, entered Jewish Hospital, April 13, 1939, complaining that in December, 1938, she noticed a tinnitus in the left ear, and double vision which persisted. Several weeks later there developed bitemporal headaches which were not incapacitating. In March, 1939, she developed nausea without vomiting. Four days prior to hospital entrance she developed pain over the left mastoid region; 24 hours later she had a high temperature with chills, dizziness and syncopal attacks.

Her past history revealed the usual childhood diseases. She had malaria at 7 years, an appendectomy at 16 years, a perineal repair at 20 years, a nasal operation and tonsillectomy and adenectomy at 20 years, and a uterine suspension at 21 years.

In 1938, she spent eight months in a sanitarium for a "nervous breakdown."

Physical Examination on Entry: Temperature, 100.5° F.; pulse, 96 min.; respiration, 18 min. A white woman lay quietly in bed and did not appear acutely ill. She was orientated in all spheres and showed no emotion or ideational disturbances. The head was normal in contour, and McEwen's sign was negative. There was tenderness on pressure over the left mastoid, with no edema or redness. There was no nuchal rigidity. The pupils were dilated and reacted to light and accommodation. The eyegrounds showed bilateral choking (see ophthalmological note below). The pharynx was negative. No tonsils were present. There was a bilateral adenopathy present in the anterior neck regions. The chest, lungs and heart were negative. The blood pressure was 105/75. The abdomen revealed a right rectus and a midline suprapubic scar. The extremities were negative. Vaginal examination was negative.

Neurosomatically: cranial nerves were negative, except the second. Muscular system showed no abnormalities. Reflexes: the corneal and gag were normal. The biceps, triceps, radioperiosteals, knee kicks and ankle jerks were all hyperactive and equal. The abdominals were present. Beevor sign was negative. There was no pathological toe signs on the right.

On the left, no reaction to stroking the sole of the foot was present. No clonus was present. Posterior column: position and vibration sense were intact bilaterally. Cerebellar signs were all negative. Sensory: on the left there was hemihypesthesia to cotton, and hemianesthesia to pinprick. Hot and cold were perceived well throughout. Special senses: diadokokinesia, stereognosis, heels to knees, fingers to nose were intact. The Romberg was negative and the gait was intact.

A lumbar puncture was done: initial pressure, 300 mm. HOH with no evidence of block. Cells: two lymphs./cmm.; Pandy was negative; Wassermann and Kahn were negative; sugar, 50 mg. per cent; protein, 23 mg. per cent; colloidal gold, flat.

An ear, nose and throat consultant gave his opinion that "this may be one of those rare cases where there is a minimum of symptoms (*e.g.*,. pneumococcus type III), and when the signs in the middle ear bone subside they leave an extensive involvement in the mastoid region, or even extending to involve the lateral sinus or brain, the latter producing the increased intracranial pressure." A caloric test was normal.

The ophthalmological consultant found the VOD, 20/30, and VOS, 20/30. The pupils were round, equal, and reacted to light and accommodation. The right disc showed an edema with +2 diopters, and the left, +3 diopters, with no hemorrhage or exudate in either. The peripheral fields were normal. The central fields showed an increase in the size of the normal blind spot.

Diplopia fields revealed that with the red glass before the right eye there was a crossed diplopia, mostly in the left horizontal field. The diplopia was noticeable chiefly in clear vision within three inches with the red glass. This, coupled with the poor reading, pointed to a definite weakness or paresis of convergence. There was very little double vision at 3 inches with the red glass.

The final opinion was that this was a bilateral choking.

The neurosurgical consultant advised a ventriculogram, which was done on April 21, 1939, and was found to be normal.

Ten days after admission it was noted that the hemianesthesia was disappearing and was gone two days later, but the choked discs still were present and had increased to +4 diopters.

The patient was discharged on April 30, 1939. Eyegrounds were not changed. The vision revealed VOD, 20/40; VOS, 20/40. Central vision slightly better, otherwise the same. No sensory abnormalities were present. An L.P. showed an initial pressure of 250 mm. HOH, otherwise normal.

During the entire interim the patient was never acutely ill, being about and engaging in the activities of the ward. Slight headache was the only subjective complaint.

Laboratory Findings: April 14, 1939: Urine negative. Fasting blood sugar, 79 mg. per cent. N.P.N., 16 mg. per cent. Blood culture was negative. Blood: W.B.C., 6,700 cmm.; R.B.C., 4,520,000 cmm. Hb. (Sahli), 86 per cent. X-ray of sinuses showed no arbnormality on the right; the left showed slight blurring of the cells in the zygomatic region. The skull plate was negative.

On May 11, 1939, the patient was readmitted, complaining of recurrence of headache, nausea, dizziness and feeling drowsy. Examination was negative except for bilateral choking of +2 diopters and left hemianesthesia to pinprick and hypesthesia to cotton. Temperature perception was intact. A lumbar puncture revealed an initial pressure of 110 mm. HOH; otherwise negative.

On May 15, 1939, the patient was discharged with no complaints. Objectively, the sensory findings had disappeared but the choking was still present. At the clinic on June 21, 1939, a complete examination was negative.

Case 2: F. H. (Reg. No. E4303) a white female, age 33 years, married, was admitted to the St. Louis City Hospital on April 27, 1939. She complained that on April 18, 1939, she developed a right frontotemporal headache which radiated to the occipital region, which persisted. On April 22, 1939, when she attempted to rise from bed she found herself too giddy to do so. For one week she had been seeing double. There were no chills but she felt feverish. For 10 days she had an earache. There was no drainage from either ear.

Past history was irrelevant except that there had been a middle ear infection in 1924, and a tonsillectomy and adenoidectomy in 1938. She had an arthritis for two years.

Menstrual periods were normal except for the last two, which were irregular. None appeared in the month of April.

Physical Examination: Temperature, 99° F.; pulse, 90 min.; respiration, 18 min. Patient was lying quietly in bed, looking acutely ill. The head was normal in contour. McEwen's sign was absent. Slight nuchal rigidity was present. Eyes: the pupils were round, equal, and reacted to light and accommodation. The fields were normal to confrontation. The right disc showed a marked choking, with no exudate. The left showed the same but to a lesser degree. Diplopia was present. A left VIth nerve paresis was present. Right ear: the right canal was clean, with no discharge. There was a large central perforation of the tympanum, with thickening of the margins, with no inflammation. Left ear: there was tenderness over the mastoid region upon pressure. The drum showed a dull redness about the superior margin but no acute inflammation. The nose showed an atrophic mucosa. The pharynx showed a slight exudate. Neck: on flexion or movement of the head to the right there was pain on the left side of the neck. Upon sitting the patient up, she complained of sensation of turning to the right. The skin showed numerous depigmented areas over the entire body. Chest, heart and lungs were negative. Extremities: there was an atrophy of the muscles of the right thigh and leg. On flexion of the right knee, the patient complained of severe pain. Blood pressure was 120/80. The abdomen was negative.

The neurological consultant found an acutely ill person, orientated and co-operative, but irritable. Somatically, there was no nuchal rigidity. A left VIth nerve weakness was present. Bilateral choked discs were present, greater on the right, without hemorrhage or exudate. All deep reflexes were present, but greater in the right upper extremity. The abdominals were absent. There was a questionable bilateral and contralateral Oppenheim and Gordon. Babinskis were negative. Other findings were normal. Detailed visual fields were impossible because of the patient's state.

The neurosurgical consultant advised an air injection, but a note on May 6, 1939, states: "This patient has definitely

improved. The VIth nerve weakness on the left has completely cleared up. Deep reflexes on the right are perhaps slightly more active than on the left. At the present time the margins of the right disc are hazy and the veins full, but the physiological cup is present. I do not think the left disc is choked at present. No evidence of C.N.S. lues. Can this be an otitic hydrocephalus which is now regressing?"

Ten days after admission the patient felt much improved, with headache quite less. She was able to sit up without dizziness. The choking was gone. The VIth nerve paresis had cleared up. On lateral gauge, there was a fine nystagmus. A caloric test was negative.

On May 24, 1939, a right radical mastoidectomy was done. There were no complications.

The patient was discharged, June 8, 1939, with no symptoms. The mastoid wound was still draining.

Laboratory Findings: April 28, 1939: Urine negative except for +2 albumin, with occasional granular casts.

May 11, 1939: Urine negative except for a trace of albumin, with occasional hyaline casts.

April 28, 1939: Blood: W.B.C., 8,400 cmm.; R.B.C., 4,500,000 cmm. Differential: Stab., three; seg., 56; lymph., 40; mono., one.

May 14, 1939: Blood: W.B.C., 8,200 cmm.; R.B.C., 4,100,000 cmm.; Hgb., 75 per cent (S).

May 1, 1939: Blood Kahn and Wassermann negative.

April 29, 1939: Blood sugar, 96 mg. per cent; blood N.P.N., 27 mg. per cent.

May 6, 1939: Lumbar puncture: Initial pressure, 180 mm. HOH; final pressure, 130 mm. HOH; two cells per cmm.; Wassermann negative; protein, 46.9 mg. per cent; culture negative.

May 12, 1939: Blood culture negative.

X-ray Findings: May 2, 1939: Skull plate negative.

May 9, 1939: Sinuses: left, negative; right showed diploic cells with a slight haziness. There was no definite evidence

of destruction. Right knee: there was a questionable infectious type of arthritis.

Temperature fluctuated between 98° and 101.5° F., with pulse corresponding. Respiration, 16 to 24 min. The mastoid at operation showed a chronically inflamed condition.

COMMENT.

Case 1: In addition to the symptoms of this entity as recorded above, some interesting observations heretofore not designated as a part of the syndrome were present: *1.* She was in an older age group than designated by Symonds. *2.* She showed a paresis in convergence. We felt that this was a secondary symptom to the increased intracranial pressure. *3.* She showed hemianesthesia to pin prick, and hemihypesthesia to touch on the left side, with the preservation of temperature discrimination. We considered these sensory findings as an added psychogenic component to the picture because of the pain and temperature dissociation. Further evidence toward this view was that this individual was a definite neuropathic type of personality in view of her history of eight months' sanitarial care for a "nervous breakdown."

It was of interest that this patient did not appear ill at any time except when she had bouts of vomiting. She made mild complaints concerning her headaches and diplopia.

Therapeutically, we found that simple pressure-relieving methods, as spinal punctures, were quite sufficient to give the necessary relief of symptoms. In this case it probably played a part.

Case 2: Here, too, in addition to the classic picture, we found that the patient was in the older age group. There was present also a subacute mastoiditis which enhanced the acuteness of the illness.

Therapeutically, she was completely relieved of her cerebral symptoms by spinal drainage only. Mastoidectomy was done to rid the patient of her original focus of toxicity.

SUMMARY.

1. Two cases of otitic hydrocephalus are reported.

2. Two new findings are reported: *a.* The incidence in an older age group; *b.* the presence of convergence paresis.

BIBLIOGRAPHY.

1. Symonds, C. P.: *Brain*, 54:55, 1931.

2. Symonds, C. P.: *Brit. Med. Jour.*, 1:53, Jan. 9, 1932.

3. Scal, J. C.: The Laryngoscope, 42:936, 1932.

4. Garland, H. G., and Seed, G. S.: *Lancet*, 1:751, Sept. 3, 1933.

5. Smith, C. H.: The Laryngoscope, 44:931, 1934.

6. Goodhart, S. P., and Savitsky, N.: *Arch. Neurol. and Psych.*, 32:1345, 1934.

7. Ersner, M. S., and Myers, D.: *Ann. Otol., Rhinol. and Laryngol.*, 45:553, 1936.

8. Bradbeer, N. H., and Gibson, P. C., Jr.: *Jour. Laryngol. and Otol.*, 52:624, 1937.

9. Williams, H. L.: *Arch. Otolaryngol.*, 25:632, 1937.

10. Kessler, M. M., and Savitsky, N.: *Mt. Sinai Hosp. Bull.* 5:486, 1938.

11. Kelly, V. B.: *Glasgow Med. Jour.*, 129:182, 1938.

4500 Olive Street.

IN MEMORIAM

AUSTIN ALBERT HAYDEN, M.D.

1881-1940.

Dr. Austin Albert Hayden was born Oct. 15, 1881, in Shullsburg, Wis.; died in Chicago of coronary thrombosis, July 10, 1940.

He received his premedical education at Creighton University of Omaha, and at the University of Chicago. He received his M.D. degree from Rush Medical College in 1904, served interneships in St. Elizabeth's Hospital and St. Anne's Hospital. He was Instructor in Ophthalmology at New York Post-Graduate Medical School, 1906-1908, and Instructor in Ophthalmology at Rush from 1908 to 1916.

He was a Fellow of the American College of Surgeons, a member of the American Otological Society, a former President of the Chicago Laryngological and Otological Society and of the Chicago Medical Society.

He was also a member of the American Academy of Ophthalmology and Oto-Laryngology, the American Laryngological, Rhinological and Otological Society, a former President of the American Association of Railway Surgeons, and a member of the Institute of Medicine and the Society of Medical History, of Chicago.

He was President of the Chicago League for the Hard-of-Hearing and of the American Federation of Leagues for the Hard-of-Hearing. In the American Medical Association he was Treasurer from 1922 to 1933, and since 1933, Secretary of the Board of Trustees.

He was an enthusiastic and devoted advocate of organized medicine, an active worker in the committee of the A.M.A. for standardizing audiometers and hearing devices, and in the later part of his career concerned himself more especially with the problems of the deafened.

To his bereaved family we offer our heartfelt sympathy.

M. A. G.

ERICH RUTTIN, M. D.

1880-1940.

News has just reached us that Prof. Erich Ruttin, of Vienna, died there Feb. 20, 1940, after a protracted illness from cardiac complications.

Dr. Ruttin was born in Prussia 60 years ago, received his medical training in Vienna and for many years was associated with the renowned Politzer Clinic there. In 1910, he was named Dozent, and in 1922, Professor Extraordinarius in Otology. Ten years later he was appointed Chief of the Ear Clinic of the Rudolf Hospital of Vienna, where he served until 1938, when he received a call from Angora, Turkey, to establish an ear clinic there.

In 1933, he visited the U. S. A. and gave special courses in Otology in various medical centres.

Many of our American colleagues have had the privilege of attending his Vienna Clinic, and many others have participated in his special courses during his American visit.

Dr. Ruttin will be remembered for his gracious personality, his teaching abilities, his wide clinical experiences, his indefatigable energy and his generous co-operation with his many loyal graduate students.

Outstanding among these contributions is his monograph on Streptococcic Mucosa in Ear Infections and his monograph on the Physiology and Pathology of the Human Labyrinth.

We extend our sincere sympathies to his bereaved family.

M. A. G.

HAROLD M. HAYS, M.D.

1880-1940.

Dr. Harold M. Hays was born in Rochester, N. Y., Sept. 26, 1880; died in Scarsdale, N. Y., of a heart attack, Aug. 21, 1940.

Graduated from Columbia University in 1902; received his M.A. degree in 1905, and got his M.D. degree from the College of Physicians and Surgeons, Columbia University.

Dr. Hays served as interne at Mt. Sinai Hospital, and was Associate Professor of Laryngology at the New York Polyclinic Medical School and Hospital from 1912 to 1915. He was an Instructor in Laryngology in the College of Physicians and Surgeons from 1912 to 1920; Assistant Otologist at the New York Eye and Ear Infirmary from 1910 to 1920; and Associate Laryngologist at City Hospital from 1910 to 1925.

He was Consulting Physician at the Sanitorium for Hebrew Children, St. Joseph Hospital and Sing Sing Prison.

During the World War he served in the A.E.F. as a Major in the Medical Corps, and became a Lieutenant-Colonel in the Medical Reserve Corps.

Dr. Hays published several books, most popular of which was his "Modern Conception of Deafness." He was a frequent contributor to the otolaryngological press, devised the electric Pharyngoscope and a Comprex Oscillator for massage of the drum membrane.

He was President of the Association of Private Hospitals, organizer of a Physicians Mutual Co-operative Association,

and President of the Park East and Park West Hospitals in New York City.

He was one of the founders and formerly President of the American Federation of Organizations for the Hard-of-Hearing, and founder, President and Director of the New York League for the Hard-of-Hearing, and was an active and effective influence in this fine field of endeavor.

He was a member of the New York Academy of Medicine, the American Medical Association, the American Academy of Ophthalmology and Oto-Laryngology, and the American Laryngological, Rhinological and Otological Association.

Dr. Hays married Miss Ethel Hallie Spear in 1907, and Miss Gertrude Elizabeth Peacock in 1934. He leaves a widow, a son by his first wife, Harold M. Hays, Jr., a brother, Arthur Garfield Hays, and a sister, Mrs. Albert L. Stern, of Baltimore.

He had a genial personality; was a dynamic, fluent speaker, participating frequently in medical discussions; possessed of considerable executive strength and generous nature.

An example of this latter characteristic was his spontaneous and generous support of the scholarship fund of Central Institute for the Deaf.

Our sincere condolence is offered to his bereaved family.

M. A. G.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

SECTION ON OTOLARYNGOLOGY.

Meeting of Feb. 23, 1940.

Nasal Accessory Sinus Disease, Types, Diagnosis and Treatment. Dr. A. C. Furstenberg (by invitation).

Dr. A. C. Furstenberg gave a most interesting talk on "Nasal Accessory Sinus Disease, Types, Diagnosis and Treatment." Lantern slides were shown.

Dr. H. I. Lillie discussed the subject.

The Surgical Treatment for Middle Ear or Conductive Deafness. Dr. Philip E. Meltzer (by invitation).

Dr. Philip E. Meltzer presented a paper entitled, "The Surgical Treatment for Middle Ear or Conductive Deafness." Lantern slides were shown.

DISCUSSION.

DR. GEORGE E. SHAMBAUGH: Dr. Meltzer has given a very complete and very beautiful review of this subject. To this I can add very little except my own experience.

I became interested in the Lempert operation when Dr. Lempert announced his first results a year and a half ago. He very kindly invited me to visit him to observe some of the operations, to see his patients and to learn the technique of the operation on the cadaver under his supervision. My first patient was operate in July, 1938, with a very satisfactory improvement in hearing. Encouraged by this result, three more patients were operated within the next two months. The fistula in the first patient then began to close, as evidenced by gradual loss of the fistula test and a loss of the hearing improvement so that five months after operation the hearing was approaching the preoperative level.

Of the first 11 patients, the fistula closed in eight, with a diminution of hearing to the preoperative level. In three cases the fistula has remained permanently open, with a positive fistula test. In one of these patients a satisfactory hearing improvement occurred, lasting four months, followed by a gradual loss to the preoperative level in spite of the fact that the fistula remained open. For some time I was mystified by this diminution in hearing but in view of subsequent observations, both on my own patients and on patients of Dr. Lempert's, I believe that bone dust which fell into the fistula during operation resulted in the new formation of bone within the fistula sufficient to obstruct the transmission of sound waves but not sufficient to interfere with the fistula test. The second of the three cases where the fistula remained open was not suitable for operation, the hearing impairment in the operated ear being the result of a nerve deafness. The mistake in diagnosis was due to failure to use masking in doing the bone conduction test. The third patient has experienced a permanent satisfactory improvement in hearing averaging 18 deb. for the conversational frequencies.

For some time I was at a loss to explain the large proportion of closures. Numerous visits were made to Dr. Lempert and as the result of observations by him and experimental work by Dr. E. P. Fowler, Jr., and others, the importance of bone dust in the closure of the fistulas has become increasingly

apparent. Since November, 1939, increased attention to meticulous removal of every particle of bone dust has resulted in the fistula remaining open in six consecutive cases, although in one of these the fistula may be closing. All of these patients except one where the fistula may be closing and where the hearing improvement averages only 10 dcb. for the conversational frequencies have experienced a satisfactory hearing improvement, as follows: 20 dcb. for the conversational frequencies; 23 dcb. for the conversational frequencies; 13 dcb. for the conversational frequencies; 20 dcb. for the conversational frequencies; and 20 dcb. for the conversational frequencies. In one of these six patients the operation was a secondary operation, where a previously closed fistula was reopened. This was the first patient operated on a year and a half ago. He now has a very satisfactory and apparently permanent restoration of practical hearing.

In conclusion, it must be pointed out that the surgical treatment of otosclerosis is still in its infancy, but for the first time it is possible to restore a substantial degree of hearing in suitable cases of otosclerosis by a one-stage surgical procedure which is devoid of risk to the patient's life and is relatively free from risk to the hearing if the technique is properly carried out.

DIRECTORY OF NATIONAL OTOLARYNGOLOGIC SOCIETIES.

American Otological Society.

President: Horace Newhart, 527 Medical Arts Bldg., Minneapolis.
Secretary-Treasurer: Dr. Isidore Friesner, 36 East 73rd St., New York.

American Academy of Ophthalmology and Otolaryngology.

President: Dr. Frank Brawley, 30 N. Michigan Ave., Chicago.
Executive Secretary: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Hotel Cleveland, Cleveland. Time: Oct. 6-11, 1940.

American Broncho-Esophagological Association.

President: Dr. Gabriel Tucker, 250 S. 18th St., Philadelphia.
Secretary: Dr. Paul Holinger, 1150 N. State St., Chicago.

American Laryngological Association.

President: Dr. Gordon Berry, 36 Pleasant St., Worcester, Mass.
Secretary: Dr. Charles J. Imperatori, 108 E. 38th St., New York.
Place: Atlantic City. Time: May 28-30, 1941.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. J. Mackenzie Brown, 1136 W. 6th St., Los Angeles.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.
Place: Los Angeles. Time: June, 1941.

Sections:

Eastern—Chairman: Dr. N. S. Weinberger, Robert Packer Hospital, Sayre, Pa.
Southern—Chairman: Dr. William G. Kennon, Doctors Bldg., Nashville.
Middle—Chairman: Dr. Walter H. Theobald, 307 N. Michigan Ave., Chicago.
Western—Chairman: Dr. Robert C. Martin, 384 Post St., San Francisco.

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology.

Chairman: Dr. LeRoy A. Schall, 243 Charles St., Boston.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia.
Place: Cleveland.